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COMMENTS ON A FOURTH YEAR OF TRAINING FOR CERTIFICATION IN OTOLARYNGOLOGY.

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BACKGROUND.

Just ten years ago, the American Board of Otolaryngology, following a review of the status of otolaryngology during and following World War II, decided the training of otolaryngologists needed strengthening. It ruled that all applicants for certification by the Board would be required to have three years of residency in otolaryngology if they began their training after July 1, 1950. At the time, there were not a few who expressed concern about this increase in the length of training. Today, however, there is no thought of a return to the former two years. There is universal acknowledgment that this move has been productive of great benefits to otolaryngology.

Ten years have passed since the announcement of this ruling. During these years, a more systematic and thorough system of appraising the residencies in otolaryngology in this country has been established. The appraisal body is known as the Residency Review Committee for Otolaryngology. It has succeeded the former Review Committee of the American Board, which, when it was in existence, passed judgment on residency training programs independent of the inspections of the American Medical Association and the American College of Surgeons. When this system was in vogue, there was

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confusion and inefficiency, and the settlement of any differences between official inspections of the three bodies (and there were some) often took one to two years to iron out. The present Residency Review Committee, then, has three cooperative sponsoring organizations: the American Medical Association, the American College of Surgeons, and the American Board. There is equal representation on the committee, four otolaryngologists from each of the three parent bodies. Each of the parent bodies has agreed to accept the judgment of these twelve men on any given residency. This means that approval or disapproval of a residency program, once it has been decided by the Residency Review Committee, has the simultaneous sanction of the American Medical Association, the American College of Surgeons, and the American Board. Order has come out of the confusion. The Residency Review Committee is more efficient, and its judgments are much more accurate and reliable. These judgments are the result of studied and often lengthy deliberations of twelve serious and dedicated men.

Over the past five years, the Residency Review Committee has processed an intense amount of data on otolaryngologic residency programs in the United States. The Committee meets twice a year, giving two days to each session. No other matters are considered save material pertaining to residency training programs in otolaryngolgy.

Good thinking and sound judgment have been used. As a result of these five years of deliberation, during which time the approval of more than a few residencies has been withdrawn (including, unfortunately, some university programs), a feeling has arisen amongst the members of this Committee that the 1948 action of the American Board of Otolaryngology, good as it was, was not enough. It has been felt that additional requirements were necessary to bolster the training of otolaryngologists. This feeling has not been crystallized by the Residency Review Committee, for this is not its function. (Approximately 50 per cent of the membership of the Residency Review Committee is made up of otolaryngologists who are not directors of the American Board of Otolaryngology.) A formalization into a regulation can properly come

only from the American Board of Otolaryngology. Such action has been taken by the Board, but it should be noted that this action did not arise out of thin air, nor was it the result of pressures by individuals or groups outside of otolaryngology. It was as a result of the combined opinions of the men who are in the best position to know the strengths and the weaknesses of Otolaryngologic Residencies throughout the country, *e.g.*, the members of the Residency Review Committee, and the men who see these same strengths and weaknesses manifest in the examination of candidates, *e.g.*, the members of the Board.

THE ACTION TAKEN.

At a meeting of the American Board of Otolaryngology held in October, 1957, in Chicago, the Board voted to add an additional year of training to the three-year training period in otolaryngology. The Board made public announcement that the additional year was to be devoted to straight surgical training.

At a subsequent meeting held in San Francisco in May, 1958, the previous action was modified. At this meeting the American Board of Otolaryngology reaffirmed the requirement of a four-year approved residency training program, at least three years of which shall be in otolaryngology. The Board modified the requirement of one year of general surgery. The additional year of training may be devoted to otolaryngology or to other fields, such as surgery, medicine, pathology, basic sciences, or research.

SOME SPECIFIC REASONS FOR THE ACTION TAKEN.

1. During the early part of this Century, some of the rightful functions of otolaryngology were lost by default. Occupied with infections of the sinuses and ears, the otolaryngologists of the country neglected to keep pace with advances made in the field of nasal and facial fractures, nasal deformities, and regional plastic surgery; also, with a few notable exceptions, the major surgery for malignancies of the sinuses, the larynx, tongue and in the neck, was not included in the training of otolaryngologists. All of this requires excellent surgical tech-

nique and sound surgical judgment, in addition to a knowledge of the physiology and function of the structures involved and an understanding of the general nutritional problems encountered.

2. In the past five years too many of the residency programs in otolaryngology have graduated men whose surgical record was deficient. (It is a requirement of the Residency Review Committee that a program under review must submit to the committee a list of the operations performed in that residency by the staff and by the residents.) The paucity of procedures in several programs has been notable. In a majority of such instances, otolaryngology has been but a division of the Department of Surgery, and these procedures were not available to the residents in otolaryngology. About 60 per cent of all the residency programs in otolaryngology are under control of the Department of Surgery in their institutions. In a goodly proportion of these, budgets are insufficient, an adequate number of hospital beds is not available, and little or no research is in progress.

3. The tempo of residency training has slowed. Many residents are married, and the end of a day's work is often 5 o'clock, and not 11 or 12 o'clock at night, as it was in former days. There is now more to teach and less time in which to do it.

4. During the past ten years, there has been an increasing number of so-called oncologic surgeons trained. These surgeons are supposedly trained to handle, surgically, malignancies in any location in the body. In the knowledge of yesteryear, this new specialist might be a boon to medicine, but in the light of modern medicine, with the necessity for knowing much about function, as well as anatomy, and with the availability of diagnostic methods and armamentaria which are best employed by specialists in their use, oncologic examination, surgery and follow-up are not equal to that which can be performed by well-trained specialists in local areas. Otolaryngologic residents must have adequate training in this field.

5. Another important reason is the action taken by the

National Institute of Health. A number of years ago this body felt that ophthalmology needed bolstering and spent some of its energies and monies for better training in ophthalmology, and the teaching of ophthalmology has improved. The National Institute of Health now feels that the teaching of otolaryngology needs strengthening; in fact, it is a specialty needing help more than most others. It is planning to spend thousands of dollars over the next ten to twenty years for the training of teachers and research men in the field of otolaryngology. This is the opinion of a group outside of otolaryngology. This is significant.

6. The most important reason for an additional year of training in otolaryngology is the recent broadening of the field. Better training is needed so that future trainees will have a working knowledge of the many facets of modern otolaryngology. The scope of otolaryngologic surgery now includes procedures which are as complicated and demanding as any in the field of surgical endeavor. (Rehabilitation service for some of these patients demands a thorough knowledge of many techniques); but there is also in this broad field the most delicate surgery of all—microscopic surgery of the ear. A very large portion of all allergy falls within the scope of otolaryngology. A modern otolaryngologist must know much about audiology. He cannot practice intelligently without this knowledge; yet, this science itself has grown to the point where studies at the doctorate level are required to encompass its broad reaches. A practical working knowledge of endocrinology is needed by the modern otolaryngologic specialist. Research in all these areas is expanding significantly. From what was thought to be a narrowing specialty in the mid-forties, otolaryngology has grown to become one of the broadest and most inclusive specialties of all. This is demonstrated when medical students are adequately exposed to it. Their response is usually one of amazement at the scope of the specialty and the opinion that they develop is at least one of respect, and often this is followed by an active interest.

HOW WILL IT WORK?

The May, 1958, action of the Board of Otolaryngology pur-

posely made the extra year of training flexible. Surgically oriented residents may spend one year of the four in straight surgery and yet receive credit for the year. Likewise, medically oriented residents may spend a year in medicine.

It may be asked, "Why should any surgery be included in the training of one who wishes to confine his practice to the medical aspects of otolaryngology?" The answer to this is that the very best internists are those who attend the surgery performed on their patients and know what is done, so they can intelligently treat them in the future. Medical otolaryngologists (they exist, and the good ones should be respected and admired) will be better specialists if they have had some surgical experience.

Various other combinations are approved. It is conceivable, for instance, that a resident may take two years of ear, nose and throat training, spend a year in otolaryngologic research, and then take a fourth year in otolaryngology. He should make a most valuable senior resident.

If the choice is a year of surgery, this year should be oriented to the needs of the otolaryngologic resident. During the year of general surgery, the individual should receive training in patient care, which would include study of fluid and electrolyte balance, wound repair, shock, flaps, grafts, and dietary deficiencies of tube fed patients. Operating room experience in the areas of interest to the otolaryngologist, namely, neck surgery, chest surgery, neurological surgery, etc., should be included. In general, it would appear advisable to place this year of surgery between the first and third years of the otolaryngologic residency, which will be after the resident has some knowledge of what should be gleaned from this extra year. This surgical experience can be obtained in the same hospital in which the sponsoring otolaryngological department functions or in a neighboring institution if it appears that better values can be realized. It is only necessary that a qualified surgeon be in charge of the approved surgical service which is to train the otolaryngologic resident.

Probably, many of the otolaryngologic residencies of the country will make this program a straight four years in

otolaryngology. Where the facilities of a department are such that the material, the teachers, and the armamentaria are available and of good standing, this will be the sensible arrangement.

A LOOK TO THE FUTURE.

The American Board of Otolaryngology has realized it must make moves which will strengthen the teaching of otolaryngology. With a long look into the future it has made the present ruling, *i.e.*, an extra year of training. Otolaryngology has expanded. There is no denying this. Almost surely, it will expand further. (The bio-acoustics of space travel, for instance.) The extra year of training has been added to prepare residents properly for the very broad field now emerging. In the future, not everyone will practice the entire field; but everyone should have training in all ramifications, so proper diagnoses, referrals, and follow-ups will be made. Only complete training will accomplish this.

Apparently, there is a need for more otolaryngologists in the United States today. One school of thought may argue for lower standards, so more practitioners will be produced. The time to increase standards is when there is a need for additional workers and when expansions in scope demand additional training. This is the situation today and is one of the reasons why the change has been made. It is believed that the response of future medical students will be adequate and rewarding.

This proposed year of additional training has seemed startling to some and to others, unnecessary. This is probably so, because not everyone is familiar with all this background material which has been available to the members of the Board. What everyone should remember is that this move has been made in view of the facts above outlined. It has been done in order to strengthen otolaryngologists and thus, to strengthen otolaryngology itself; all other reasons are subservient to this.

POSTMORTEM CHANGES AND ARTIFACTS IN HUMAN TEMPORAL BONES.*†

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I. INTRODUCTION.

The recognition of autolytic changes in human specimens is particularly important because, as a rule, the temporal bones are removed and fixed some hours after death, when autolysis is well under way. This process may have developed to the point where pathologic change is masked or its recognition impeded; furthermore, autolysis and pathologic change often are intermingled with artifacts, so that a differentiation between one and the others remains uncertain. To minimize these complications, the technique of injecting a fixative into the middle ear soon after death was developed. The rationale was that the round window and probably the oval window are the main pathways of the fixative. Then, if this was injected into the middle ear soon after death there was a good chance to eliminate or minimize the autolytic process. Another procedure based on the same reasoning was removal of stapes before immersion.

The purpose of the investigation to be described here was to compare the results in human temporal bones treated by three procedures—removal of stapes before immersion, immersion, and early fixation by injecting formalin into the middle ear soon after death. The investigation was concerned with the preservation of the inner ear structures, study of patterns of postmortem changes and artifacts in human temporal bones, and comparison with those observed in guinea pigs.[1]

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II. MATERIAL.

The study included 53 human temporal bones taken from 28 cadavers which were kept under refrigeration (between −1.1° and 4.4° C) until autopsy. The technique for removing human temporal bones as described elsewhere[2] was used.

Removal of the stapes before immersion in fixative was done in two specimens. Helly's fluid was used as the fixative and nitric acid as the decalcifier. The immersion procedure, which consisted of removal of temporal bones and their immersion in fixative, was employed in 25 specimens. In this group Helly's fluid, Zenker's solution, or Heidenhain-Susa were used as fixatives, and nitric acid, trichloroacetic acid, or formic acid plus sodium formate, as decalcifiers. The technique for injecting fixative into the middle ear, as developed in the Department of Otolaryngology of the University of Chicago Clinics, was as follows: One injection (10 cc.) of 10 or 20 per cent formalin through the tympanic membrane, as soon as possible after death; removal of temporal bones at autopsy and immersion in fixative. This group included 26 specimens. Fixatives and decalcifiers as in immersion procedure were employed.

The temporal bones were sectioned at 20 microns, and one of every ten or twenty sections stained as described elsewhere[3] with hematoxylin-eosin or Mallory's azan. The preparations were studied under regular light microscopy.

III. DEFINITIONS.

There are two types of artifact which should be differentiated: one is primary, and is the result of interaction between reagents and membranes, fluids and cell constituents. In the guinea pig this result was called the normal microscopic cytoarchitecture of the inner ear[1] when fixation by intravital perfusion of Heidenhain-Susa and staining with hematoxylin-eosin were used.

The normal microscopic cytoarchitecture of the human inner ear must be defined in terms of the best result obtained by any procedure. Examination of these specimens showed a

homogeneous rather than a granular-like cytoplasm, in almost all cell populations of non-nervous tissue. Undoubtedly this, together with some precipitates and globular formation, are signs of postmortem changes. Golgi's apparatus, centriole and mitochondria were absent, as they were in animal specimens. In the nucleus, the granular-like formation was also absent,

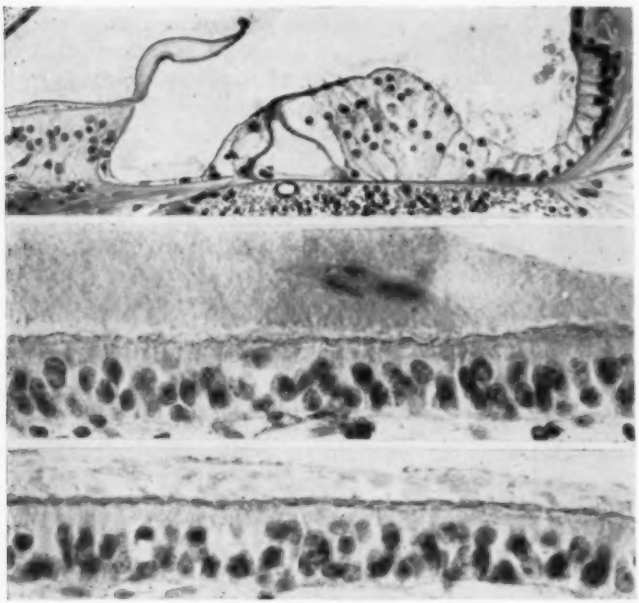


Fig. 1. Good preservation of structures with minimum postmortem changes. From top to bottom, Organ of Corti (H-E, X255), macula saccularis (H-E, X625) and macula utricularis (H-E, X625). Fine details of cell constituents can be identified.

and the kariosomes barely differentiated. The plasmosome stained more prominently than any other cell constituent. The cell bodies of Corti's ganglion presented swelling and homogenization, while Scarpa's ganglion was better preserved. Size, shape and position of cells and membranes were, in general, comparable to those found in animals. Any change

in this picture, other than pathologic or autolytic processes, was called secondary artifact. For convenience, the term artifact has been used here to mean, unless otherwise specified, the secondary artifact.

In the series of 53 human temporal bones a wide variation in degree of preservation was found. The results were defined in terms of the efficiency of the methods in preserving

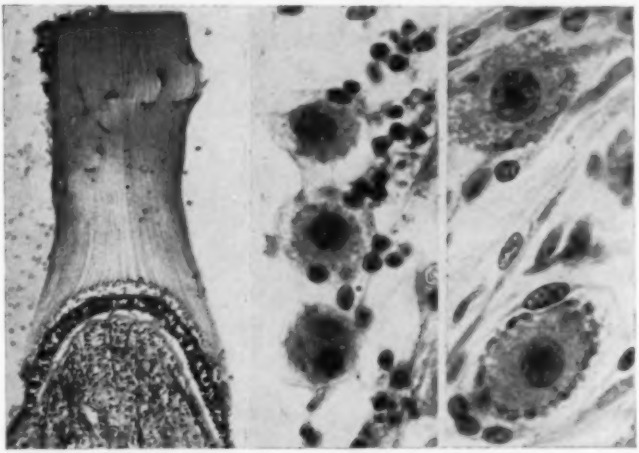


Fig. 2. Good preservation of structures. Left, crista and cupula of a semicircular canal (H-E, X105), in which there is some precipitate around the cupula and also over the surface of sensory epithelium. Center, cell bodies of Corti's ganglion (H-E, X700) showing edema and hemogenization but in general nuclei are well preserved. Right, cell bodies of Scarpa's ganglion (H-E, X700) in which the fine details of cell constituents are preserved.

the inner ear structures from postmortem changes and artifacts. Using this criterion, the specimens were divided into four categories: *1.* Good; postmortem changes and artifacts were at a minimum. These specimens were considered to demonstrate the normal microscopic cytoarchitecture of the inner ear and they were the ones to which all others were compared. In Figs. 1 and 2 the best samples of structures ideally preserved are presented. The degree of preservation for individual specimens, in general, varied from one structure

to another; the variation consisted mostly in more or less advanced postmortem changes. *2.* Fair; still more advanced postmortem changes and/or artifacts. Precipitates and distortion of cells by swelling and homogenization were the most prominent features; however, cell membranes, nucleus and general cytoarchitecture were preserved, and consequently the specimens were useful for histologic studies. *3.* Poor; the cells were so distorted by postmortem changes and artifacts that usefulness of these specimens for histologic studies was questionable. The distortion affected the organ of Corti more than structures of vestibular receptors. Finally *4.* unsatisfactory; useless for histologic studies of fine details.

It must be clearly understood that this grouping of results into four categories was based only on the efficiency of the procedure for preserving the inner ear. Any specimen, no matter how distorted it is by advanced autolytic process or artifacts, can be used for histologic studies of gross pathologic changes. In cases of tumor, hemorrhage, atrophy of cochlear and vestibular nerve, infection, and other conditions the lesion can be identified, even though the fine details may be completely lost. In other cases, such as acoustic trauma, intoxication and degenerative processes, the preservation of fine details is critical. Our grouping was made with this latter type of pathologic change in mind.

For convenience, the following definitions are given. Injection time was defined as the time between death and injection of formalin into the middle ear. Pre-refrigeration time meant the time between death and transfer of cadaver to the Pathology Department, as was found in the medical record. We were informed that all cadavers are put in the refrigerator as soon as received. In a few cases the pre-refrigeration time was questionable because it was too short. Refrigeration was defined as the time the cadaver remained in the refrigerator, and immersion time as the time interval between death and immersion of the specimen in fixative.

IV. PRESERVATION AFTER REMOVING THE STAPES.

In one cadaver the pre-refrigeration time, refrigeration and immersion time were three, eight and 13 hours respec-

tively. The stapes was removed in both ears, just before immersion. The preservation of both temporal bones was classified as fair. The Organ of Corti presented edema and homogenization of cells, precipitates, and globular formation. In one specimen Reissner's membrane collapsed over the Organ of Corti without compressing it; in the other, Reissner's

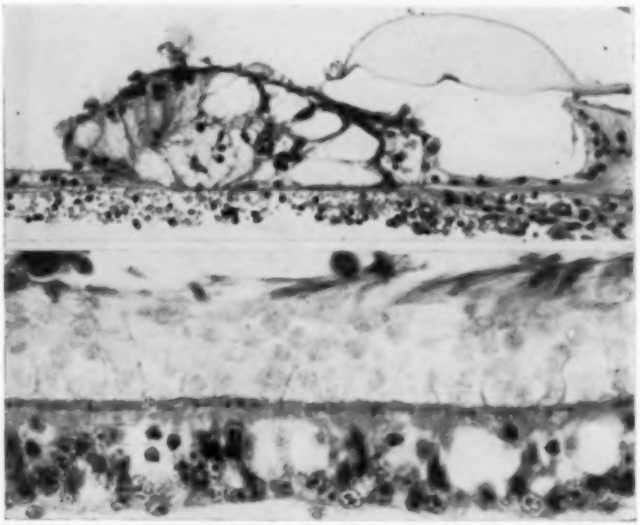


Fig. 3. Fair results obtained by removing the stapes before immersion of the specimen in fixative. H.H., 58 years, cancer of the lung. The Organ of Corti (H-E, X255) presents edema of cells, precipitates and globules, but cells can still be identified. The macula sacularis (H-E, X625) shows edema of some sensory cells, precipitates and globules between otoconia and sensory epithelium.

membrane was straight. The sensory epithelium of vestibular receptors showed the same pattern of postmortem changes and similarly, the vestibular walls collapsed in one specimen without compressing the receptors. In Fig. 3 the Organ of Corti and a region of macula sacularis are illustrated. The ganglion of Corti showed swelling and homogenization of cell bodies but no pyknosis. In Scarpa's ganglion the granular-

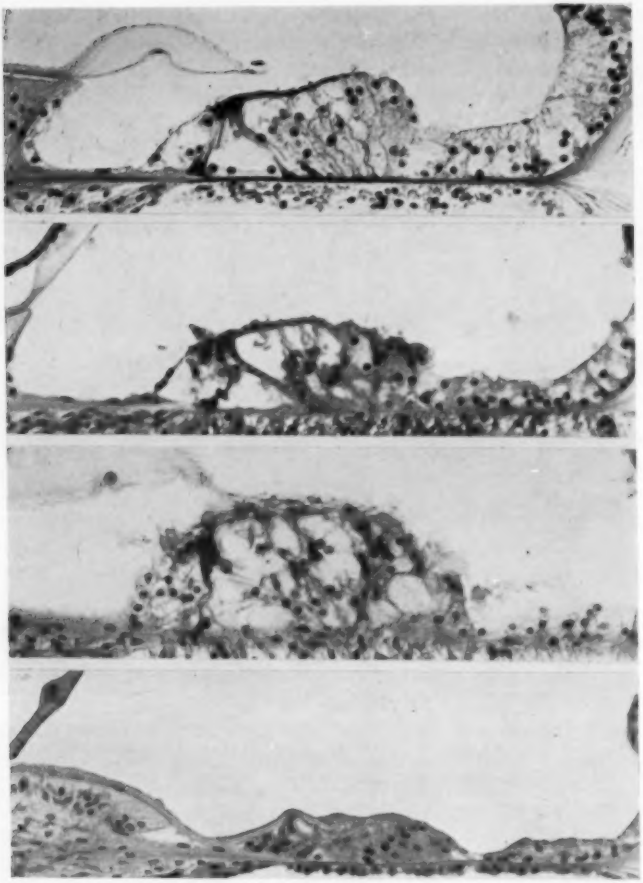


Fig. 4. Type of results in the Organ of Corti obtained with the immersion procedure. From top to bottom, good, (Mallory's azan stain, X255); fair, (H-E, X255); poor, (Mallory's azan stain, X255); unsatisfactory (H-E, X255). Fair and poor results were caused mainly by postmortem changes while the unsatisfactory was due to compression of the Organ of Corti.

like cytoplasm and constitutents of the nucleus were differentiated.

The procedure gave results which were no better than those of other methods. It had the disadvantage that structures of middle ear may be largely destroyed during removal of stapes, and consequently, the procedure is not suitable for these particular studies. Investigations with guinea pigs[1] showed that preservation of cochlear structures was as good by immersion procedure alone as it was by removal of the stapes. With the latter, vestibular structures were better preserved, but there was a tendency to produce collapse of the membranous labyrinthine walls.

Recapitulating, the investigation in animals and the study of two human temporal bones indicates that the removal of the stapes had more disadvantages than simple immersion.

V. PRESERVATION AFTER IMMERSION PROCEDURE.

The histologic picture of the cochlea, vestibule and nervous elements will be described and discussed separately.

Cochlear Receptors. The results were as follows: Good in seven, fair in two, poor in five, and unsatisfactory in eleven. The four types of preservation are illustrated in Fig. 4. Fair and poor results were brought about mainly by postmortem changes, while preparations were judged to be unsatisfactory primarily because of artifacts.

Different degrees of swelling and homogenization with or without pyknosis characterized the picture of postmortem changes. In poor results, large and numerous space formations in the Organ of Corti were seen, but we could not decide whether this represented edema of cells or intercellular spaces. Precipitates and globular formation over surfaces, in Organ of Corti and/or cochlear spaces were consistently observed. Comparison of these findings with those of experimental animals showed that the characteristic sequence of autolysis as a function of time was not seen in human as clearly as in animal specimens. Fig. 5 shows that the progress of autolysis was delayed in human preparations. The immersion time in

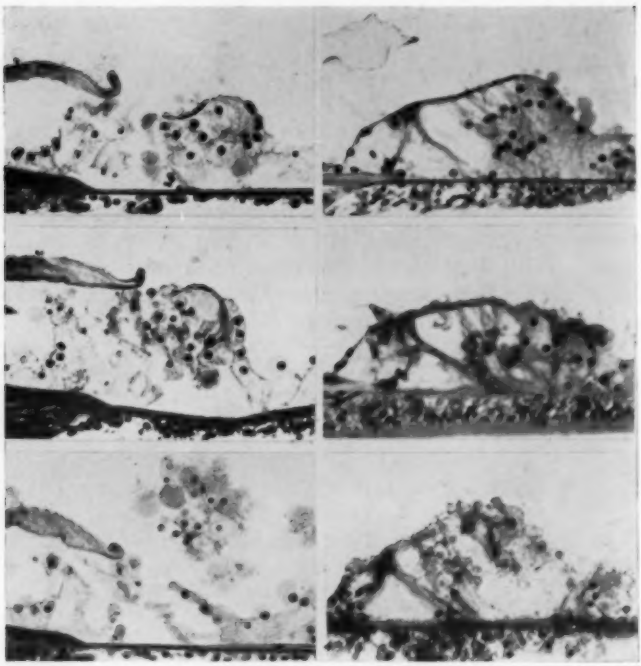


Fig. 5. Comparison of autolytic changes in the Organ of Corti as function of time in guinea pig and human specimens. Immersion procedure was used in all these cases. The photomicrographs in the left column belong to guinea pig specimens immersed in fixative 6, 10 and 20 hours after death (from top to bottom). In the right column are the corresponding samples of human specimens immersed 6, 11 and 18 hours after death. The figure illustrates that the autolytic process was faster in animal than human specimens. The difference may be due primarily to refrigeration of human cadavers. Mallory's azan stain was used in these sections, except the middle one in the right column, which was stained with H-E. All photomicrographs X255.

the 25 specimens varied between four and 20 hours. In general, the degree of postmortem change increased as this time was prolonged. Occasionally a specimen fixed a few hours after death presented more advanced autolysis than another which had been put into fixative many hours later. Disintegration of cochlear structures, as seen in later stages of the autolytic process in animals, was found only in the stria vascularis of one specimen. This was a child of 15 months

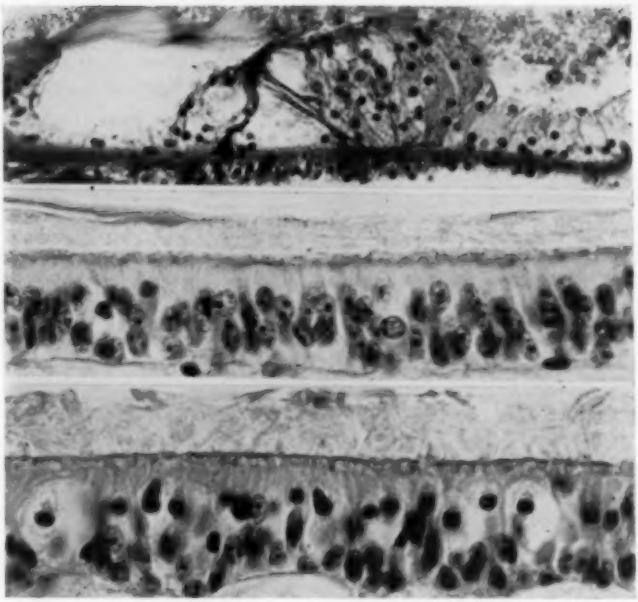


Fig. 6. Good preservation of structures in a specimen in which the cochlea was broken during dissection of temporal bones. By mistake, the specimen remained in decalcifier for about four months. From top to bottom, Organ of Corti (Mallory's azan stain, X255), which presents precipitates over the surface; macula sacculari s (H-E, X625) where no postmortem changes are detectable, and macula utricularis (H-E, X625) in which some sensory cells present edema and pyknosis. The good preservation of structures might be attributed to facilitation of penetration of fixative through the broken cochlea; however, in the opposite ear, where the cochlea was not broken, the same results were obtained. Refrigeration of cadaver seems to be the main factor in preserving this specimen from progress of autolysis.

with a fracture of the base of the skull. Immersion time of these temporal bones was 12 hours. Disintegration of the stria vascularis was similar to that found in animal specimens immersed ten hours after death.

The reason for the difference in the trend of postmortem changes in human and animal preparations was not clear. We did not find evidence which could be related to terminal temperature of the patient, fixatives, decalcifiers or other steps in the histologic technique. Difference in species and

conservation of cadavers in refrigeration until autopsy seem to be the most probable factors. Although refrigeration delays the autolytic process, the pre-refrigeration time must be considered, because the cadavers were exposed to room temperature (about 25.5° C) and consequently autolysis progressed steadily. This time varied from 30 to 170 minutes. As expected, the best results were found in specimens with the shortest pre-refrigeration time, but even among these there were unsatisfactory preparations. An example which may illustrate the importance of early refrigeration is given in Fig. 6. This was removed from a cadaver refrigerated 30 minutes after death and the temporal bones immersed in fixative 14 hours later. The photomicrograph shown in Fig. 6 is of the ear in which the cochlea was broken during dissection. This temporal bone was misplaced during decalcification, where it remained for about four months. The good results might have been attributed to facilitation in penetration of fixative; however, the opposite ear, where the cochlea was not broken, and the temporal bone not misplaced, showed as good results. This suggested that early refrigeration of cadavers is very likely of considerable importance in preventing or impeding postmortem change in temporal bones.

Detachment of tectorial membrane from the Organ of Corti was a consistent finding, but the membrane did not shrink as much as in experimental animals. This may be a matter of difference in size of this structure in the two specimens. Detachment of this membrane from the limbus was also observed in cases with advanced postmortem changes. The position of Reissner's membrane varied from straight to complete collapse (see Table I). Artifacts, such as detachment of Hensen's from Deiter's cells, space formation in internal and/or external sulcus cells, were almost absent.

Compression of the Organ of Corti accounted for almost all the unsatisfactory results. This phenomenon was characterized by collapse of the structures to such an extent that cells frequently could not be identified. The cytoplasm was dense, the nuclei were pyknotic and cell borders diffuse. The pillars of Corti and cuticular lamina collapsed so that the tunnel of Corti and Nuels' spaces disappeared. As a rule the tectorial

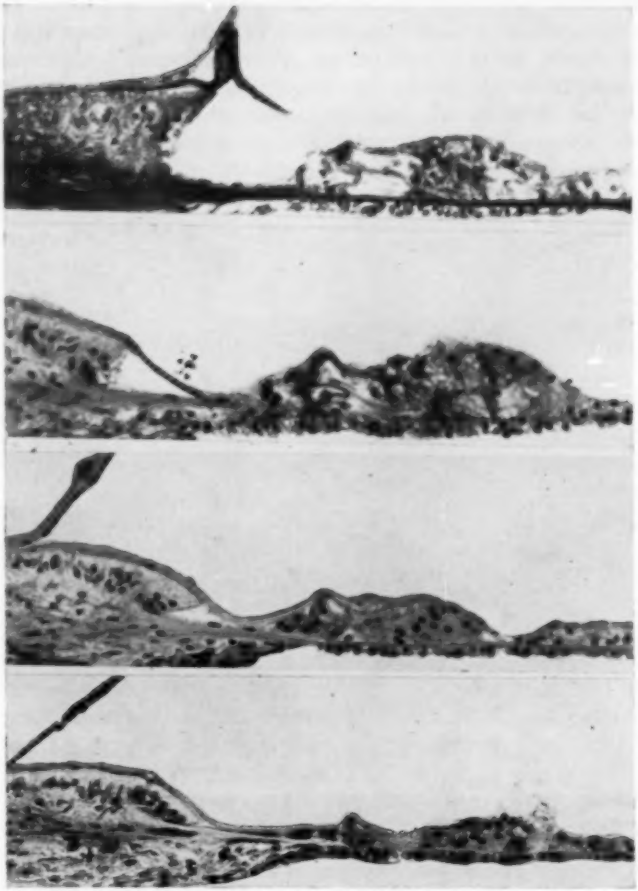


Fig. 7. Several degrees of compression of the Organ of Corti, from slight distortion to complete flattening. In all cases the tectorial membrane is reduced to a thin lamina which generally appears collapsed over internal sulcus cells and Organ of Corti. The degree of compression is reflected also in the limbus, which in extreme cases may be flattened over the internal sulcus, as the photomicrograph at the bottom of the figure illustrates. In all cases, the mesothelial cells appear agglutinated to the basilar membrane. The section at the bottom of figure was stained with H-E; the others with Mallory's azan stain. All photomicrographs X255.

membrane was flattened to a thin lamina, which either collapsed over internal sulcus cells and Organ of Corti (most of the cases), or it was detached from it and agglutinated to Reissner's membrane or the limbus. Several degrees of compression were found, from moderate distortion to flattened structures. A few examples are illustrated in Fig. 7. As this figure shows, the limbus also presented several degrees of

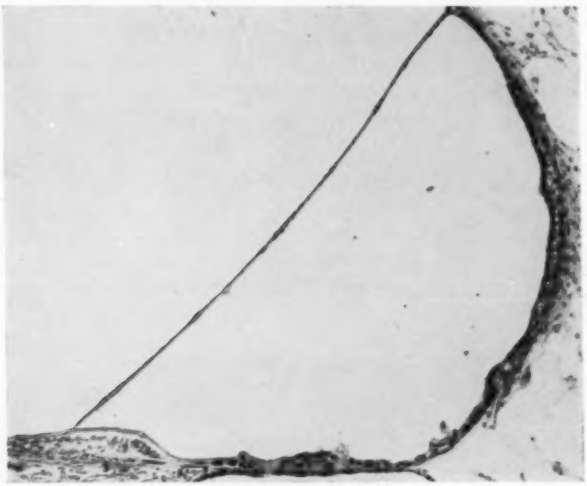


Fig. 8. This photomicrograph of the scala media illustrates the neatness of the section in a case of compression of the Organ of Corti. Notice the clear cut border of epithelium surfaces and absence of precipitates and/or globules. H-E, X120.

flattening. The absence of precipitates and globular formation was characteristic, and gave to the sections an aspect of neatness (see Fig. 8) not found even in the best preserved specimens; however, using Mallory's azan stain and observing at high magnification, sometimes a thin layer of agglutinated precipitates could be seen over the Organ of Corti and stria vascularis. Surface of the stria vascularis was unusually clear-cut and its flattened epithelium presented many pyknotic nuclei. Reissner's membrane occasionally was broken; in

other cases it may be collapsed over the Organ of Corti. In all the cases mesothelial cells were agglutinated to the basilar membrane. Compression was also visible in the vestibular system where the otoconia agglutinated over the maculae and sometimes the cupulae over the cristae. In these structures the height of sensory epithelium seems to be diminished. As in the cochlea, the vestibular labyrinth did not present precipitates and globular formation.

The cause of the compression phenomenon has been the center of several theories and debates. According to Witmaack[4,5] perilymph is produced by a process of filtration, while endolymph is formed by a secretory process which takes place in the stria vascularis, Organ of Corti, cristae, maculae and its marginal epithelium. Wittmaack claimed that endolymphatic pressure must be higher than perilymphatic, otherwise the architecture of the cochlear duct could not be maintained. Under some circumstances the endolymphatic pressure may be diminished or the perilymphatic pressure may be increased. In either case, Reissner's membrane collapses over the Organ of Corti, which will be affected in varying degree, from mild distortion to complete flattening. This picture was called "hypotonic degeneration" by Wittmaack, who claimed it is essentially a vital process. Mygind[6] stated that perilymph is a filtration product mainly of the vestibular region of spiral ligament while endolymph is secreted mainly by the stria vescularis. Normally both mechanisms are in balance. Mygind found the picture described as hypotonic degeneration by Wittmaack only in animals fixed by intravital perfusion. He assumed that because of some caprice of nervous mechanism, filtration predominated over secretion during perfusion of saline. Consequently, Reissner's membrane collapsed over the Organ of Corti, causing several degrees of compression. He called this picture "endolymphatic compression" and, like Wittmaack, claimed it is a vital process. Mygind[6] also stated that in human temporal bones the process of autolytic dissolution may induce collapse of the Organ of Corti so that it may be confused with the true compression as previously described.

In either theory the factor which brings about the com-

pression phenomenon in human or animal specimens seems to be mainly the pressure applied over the Organ of Corti. We think, as has been maintained in the past,[7] that the phenomenon of compression is an artifact. The arguments which lead to this conclusion were as follows:

1. Békésy[8] demonstrated that the tectorial membrane and reticular membrane are stiff plates; furthermore, Katsuki and Covell[9] also demonstrated that the reticular membrane with attached pillar cells is particularly resistant to being broken. They stated, "The rather firm anchorage of the upper ends of the internal and external hair cells in the reticular membrane and the obviously rigid support of the phalangeal cells and pillar cells tend to produce a unit that is compact and not readily broken up." These observations suggested that a high pressure, which is not likely to be produced in the living state, would be necessary to compress the Organ of Corti. It may be argued that a loss in turgescence of cells, as put forward by Wittmaack,[10] might facilitate the collapse of the unit. But Katsuki and Covell[9] also found that the body of hair cells can be teased out of the Organ of Corti, leaving their cuticular border with the hairs and reticular membrane intact. Schuknecht[11] illustrated in a cat with presbycusis that the frame formed by internal pillar cells and reticular membrane remained in its normal position, even though the external pillar cells and hair cells were absent. These observations have shown that large changes in sensory and supporting structures of the Organ of Corti can take place without much altering the frame work formed by reticular membrane and pillar cells. This argument supported the theory that the compression phenomenon seems to be related to changes which take place during the histological treatment of temporal bones.

2. To explain collapse of the limbus (see Fig. 7) by a vital process is still more difficult. The limbus is a compact structure made of thick fibers, connective tissue cells and ground substance. and it is widely attached to the spiral lamina. Studies on presbycusis[11] and acoustic trauma[12] have shown that the connective tissue cells may disappear, but the frame formed by the fibrous tissue remained as in the normal

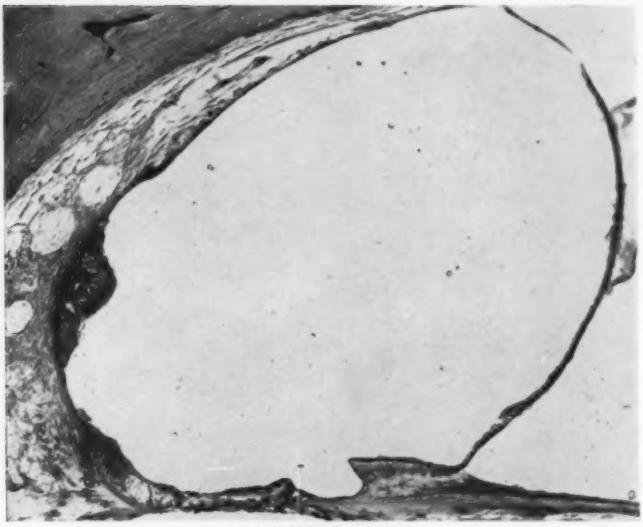
animals. These investigations showed that the limbus is particularly resistant to distortion even in the presence of degenerative processes. Explanation of the collapse of this structure, as seen in human specimens, by pressure, degeneration, or another mechanism except artifact, is unsatisfactory.

3. The absence of precipitates and/or globular formation is very difficult to attribute to any part of a vital process. In the best preserved human specimen, and in the best animal sections prepared by intravital perfusion,[1] some debris always was found over the epithelial surfaces or in the scalae. The amount of debris increased as autolysis progressed or when pathologic changes were present. For these reasons and because the preparations with compression of the organ of Corti are too neat, we doubt this feature of the phenomenon can be produced by a mechanism other than artifact.

4. Although the mechanism of dilatation of cochlear duct in Ménière's disease remains unexplained, we may assume that an overpressure probably was developed; but the evidence seemed to indicate that this overpressure is not enough to produce collapse of the Organ of Corti, tectorial membrane and limbus.

The reports on histopathology of Ménière's disease,[13] where dilatation of cochlear duct was demonstrated, showed degeneration of the Organ of Corti, but not the features of compression. Day and Lindsay[14] reported a case of Ménière's disease of the right ear in which electrocoagulation of the vestibule was done. The histologic study of the temporal bone demonstrated dilatation of cochlear duct and a flat Organ of Corti showing some features in common with the compression phenomenon, as Fig. 9 illustrates.

These changes, which seemed to be degenerative, are incompatible with function, as shown by the audiogram, and the question was raised whether they represented an artifact or a pathologic change. Fig. 10 illustrates a case taken from our series, in which there was dilatation of cochlear duct without compression of the Organ of Corti and hearing loss as the audiogram indicates.



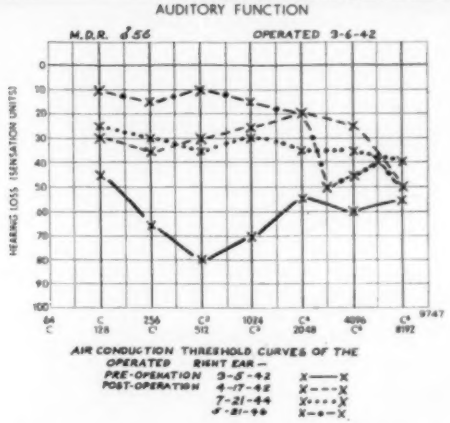


Fig. 9. Organ of Corti of a case of Meniere's disease, which was treated by electrocoagulation of the vestibule. Dilatation of cochlear duct was found throughout. The changes in Organ of Corti are incompatible with auditory function. The picture presents the features of compression, and the question was raised whether the changes are artifacts or pathologic changes. H-E, X115. (Courtesy Dr. J. R. Lindsay).

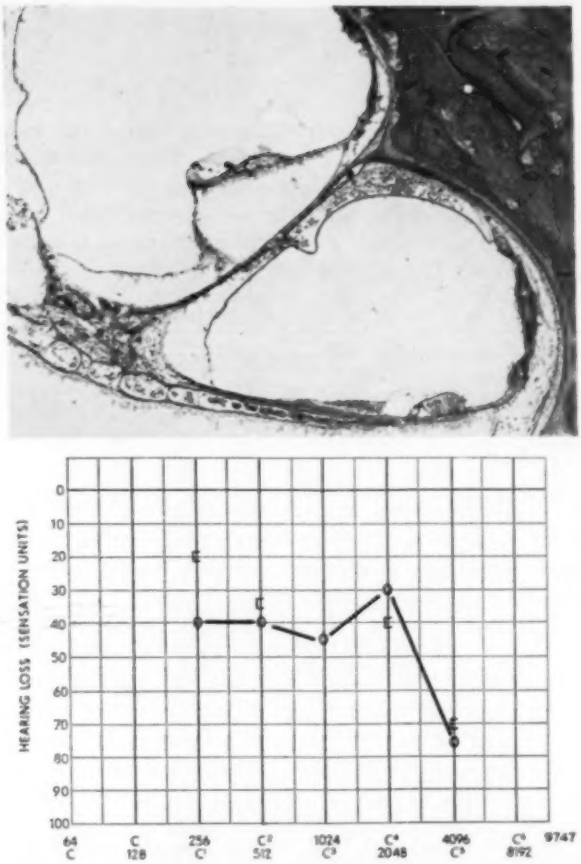


Fig. 10. Dilatation of cochlear duct without compression of the Organ of Corti. O.H., 66 years; ruptured aortic aneurysm. The photomicrograph shows the apical turns (Mallory's azan stain, X38). Throughout the cochlea no signs of compression were found, in spite of pronounced dilatation of cochlear duct. Notice that tectorial membrane, limbus and Organ of Corti retain their shape, and also that precipitates and globular formation are present. The audiogram was taken 20 days before death.

5. If the overproduction of perilymphatic fluid or diminution of endolymphatic fluid brings about the compression, then Reissner's membrane should be found collapsed over the Organ of Corti. In Table I the position of the membrane for the 53 specimens is presented. Included in Column 1 were all the cases in which the membrane was straight or deviated in one way or the other. In Column 2 were those in which the collapse reached only the limbus; in Column 3, all the cases where Reissner's membrane touched the Organ of Corti. In most of these cases, the collapse was restricted in extent mainly to the second turn. Included in Column 4 were cases

TABLE I. POSITION OF REISSNER'S MEMBRANE.

		1.	2.	3.	4.
Results	No. Cases	Normal	Collapsed over Limbus	Collapsed over Organ of Corti	Not Classified
Good	18	9	2	6	1
Fair	7	2	1	4	
Poor	6	2	2	2	
Unsatisfactory	22	4	2	13	3

in which dilatation of cochlear duct or major distortion of the cochlea by artifacts made it impossible to include them in the other categories. In cases with compression of the Organ of Corti (unsatisfactory results) the tendency of Reissner's membrane was to collapse, but the same picture also was found in cases without compression; furthermore, some specimens with compression presented a straight membrane. Evidently, collapse of Reissner's membrane can not be used as an indication of overproduction of perilymph or diminution of endolymph. The theory of Mygind[6] about the position of Reissner's membrane and endolymphatic compression is not applicable to human temporal bones because only postmortem immersion is used. Since the compression, as seen in human material, was similar to that reported by Mygind in animals fixed by intravital perfusion, the theory that it was brought about by perfusion is questionable.

6. If compression is related to pathology or other disturbances of the inner ear, then comparison of the histologic pic-

ture and hearing tests may give some basis for that relationship. Audiograms were available for 29 ears. In some, the testing was done a few days before death, in others at varying intervals during the year preceding expiration. In Table II the results are summarized.

Seven of 15 patients with normal hearing, (see Column 1) as determined by the averaging method, presented compression. Because these results were not correlative to function (see Fig. 11), we believe the compression phenomenon is an artifact. It is probable that some diseases of the inner ear present some features of compression, but the relationship of the disease to the histologic findings still must be demon-

TABLE II.

HEARING LOSS AS DETERMINED BY THE AVERAGING METHOD.

Results	No.	0-10 db.	11-20 db.	21-30 db.	31-40 db.	41-50 db.	51-60 db.
Good	10	5	2	1	2		
Fair	2	1			1		
Poor	2	2					
Unsatisfactory	15	7	4	2			2
Total	29	15	6	3	3		2

strated. On the other hand, we believe that several times in the past the compression phenomenon has not been identified, or has been confused with pathologic changes. For instance, Bunch and Wolff[15] reported three cases of degeneration of the Organ of Corti incompatible with function. As far as we can judge from the reproductions, the Organ of Corti presented the features of the compression phenomenon. Similarly, Ozaki[16] illustrated a case of streptomycin ototoxicity which had all the characteristics of compression. Reissner's membrane appeared to be absent; probably it was broken and agglutinated over surfaces of the cochlear duct. Organ of Corti and tectorial membrane were collapsed and the limbus presented a moderate flattening. No precipitates nor globular formation could be detected.

7. The suggestion of Mygind[6] that postmortem changes may induce endolymphatic compression in human specimens is questionable, since the trend of autolytic process is just the

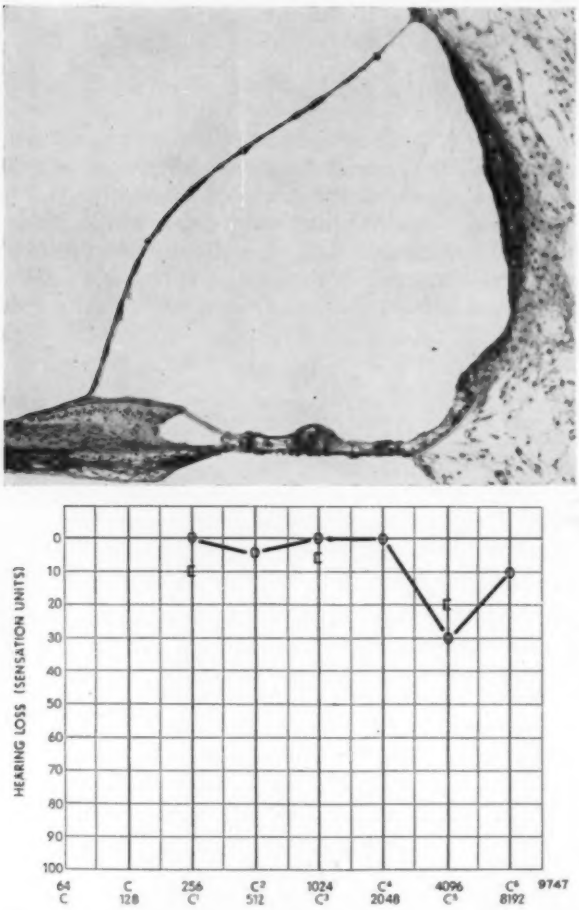


Fig. 11. The figure illustrates a case of compression of the Organ of Corti, in which the changes are incompatible with auditory function. E.G., 47 years, cancer of the tongue. The photomicrograph (Mallory's stain, X110) shows all the features of compression. The audiogram was taken four months before death.

opposite, *i.e.* to produce precipitates, globular formation and disintegration of cells.

8. If compression is a vital process, then we would expect to find it in both temporal bones rather than on one side only. Of the 25 subjects in which both temporal bones were saved, compression was found to be unilateral in 13 and bilateral in four; furthermore, unilateral compression was accompanied in many cases (8 subjects) by good results in the opposite ear, as Fig. 13 illustrates (see legend).

9. The agglutination of mesothelial cells to the basilar membrane, found only in cases of compression, indicates that the phenomenon also took place in scala tympani. This finding, and the fact that round window membrane did not show signs of pressure, suggested that the agglutination of mesothelial cells to basilar membrane is related more to histologic treatment than to pressure or a vital phenomenon.

All these considerations suggested that compression phenomenon is not related to a vital process, as Wittmaack[4,5] and Mygind[6] contended. The evidence pointed more to artifacts. In the long process of histologic preparations many errors in the technique may be committed, or the interaction between reagents and delicate structures may be different from those expected, and thus marked changes in the cytoarchitecture can be produced. In this series, there was no evidence of what kind of error may be committed and no indication as to where in the histologic technique an error may be critical.

The arguments supported the theory that compression is an artifact. Any effort to prove or disprove this theory is to be desired, because it is of considerable consequence to know whether the phenomenon is an artifact or a vital process.

Vestibular Receptors. Postmortem changes in the vestibular receptors paralleled those in the cochlea. Swelling and pyknosis of sensory cells were the most prominent features. In many cases, sensory and supporting cells of margins of maculae were better preserved than the center. This may be due to penetrating characteristics of fixatives. Precipitates and globular formation were usually found between otoconia

and maculae, and also between cupulae and cristae. Advanced postmortem changes, as seen in later stages of autolytic process in animals were rare. As previously described, compression phenomenon was also detected in the vestibule; however, due to the simplicity of the cytoarchitecture of vestibular receptors, the distortion was moderate relative to that seen in the Organ of Corti.

Neural Elements. Ganglion of Scarpa was better preserved than the ganglion of Corti, as had been anticipated.[1] The degree of postmortem change was not as might have been expected, however. In animals the neural elements were the first among all structures of the inner ear to show signs of autolysis. Swelling, homogenization, pyknosis, and finally disintegration, was the characteristic pattern. In human specimens the picture was different. The cell bodies of ganglion of Corti presented swelling and homogenization, pyknosis in a few cases, but no disintegration. This probably was due to the fact that autolysis was delayed by refrigeration of cadavers. In the ganglion of Scarpa the degree of preservation as a function of time was such that not much difference was seen from one specimen to another. Fig. 12 illustrates the state of both ganglions as a function of time. Two main factors seemed to be involved in the good preservation of the ganglion of Scarpa; one was refrigeration; the other, anatomical arrangement. Scarpa's ganglion is in the inner meatus and consequently, readily exposed to fixative.

VI. PRESERVATION AFTER INJECTION OF FIXATIVE IN THE MIDDLE EAR.

This group included 26 specimens. In 14 cases the injection time was 15 to 45 minutes, and in 12 cases was 70 to 120 minutes. Pre-refrigeration, refrigeration and immersion times were about the same as in immersion procedure. The results were classified as: 11 good, three fair, one poor and 11 unsatisfactory.

Cochlear Receptors. The four types of preservation in the Organ of Corti are illustrated in Fig. 13. Comparison with the result of immersion procedure showed that for either one

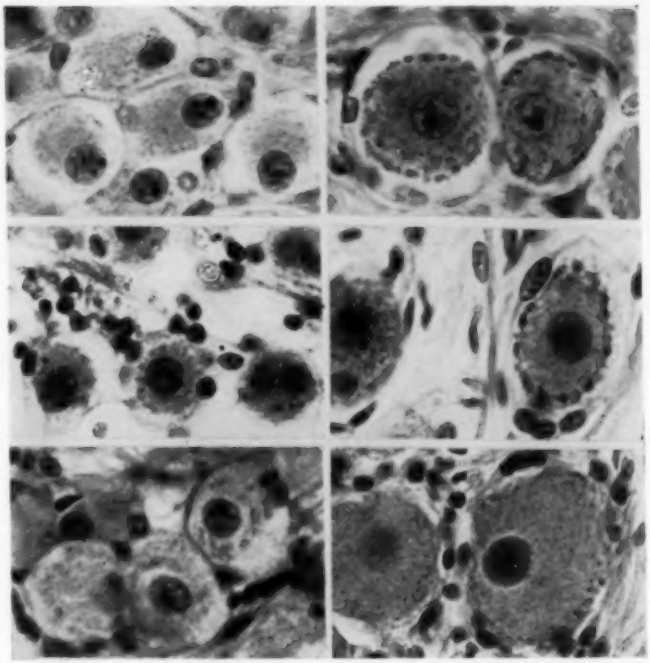


Fig. 12. Autolytic changes in ganglions of Corti and Scarpa as function of time found in specimens fixed by immersion procedure. From top to bottom, on the left, cell bodies of Corti's ganglion of specimens immersed in fixative 7 (X700), 10 (X700) and 20 hours (X625); edema and homogenization are the most prominent features; the nuclei are preserved although some may be pyknotic. On the right are the corresponding cell bodies of Scarpa's ganglion (X625, X700, and X500 respectively), in which the cell constituents are better preserved than in Corti's ganglion. All the sections from which the photomicrographs were taken were stained with H-E.

the degree of preservation was similar (see Fig. 4). From this point of view there was no advantage of one procedure or the other; however, injection of fixative into the middle ear soon after death gave a higher proportion of well-preserved specimens than was obtained by immersion alone. Early fixation helped in preserving the inner ear structures from postmortem changes, but refrigeration still seemed to be the main factor. Three observations supported this assumption: one, the best results from immersion and injection

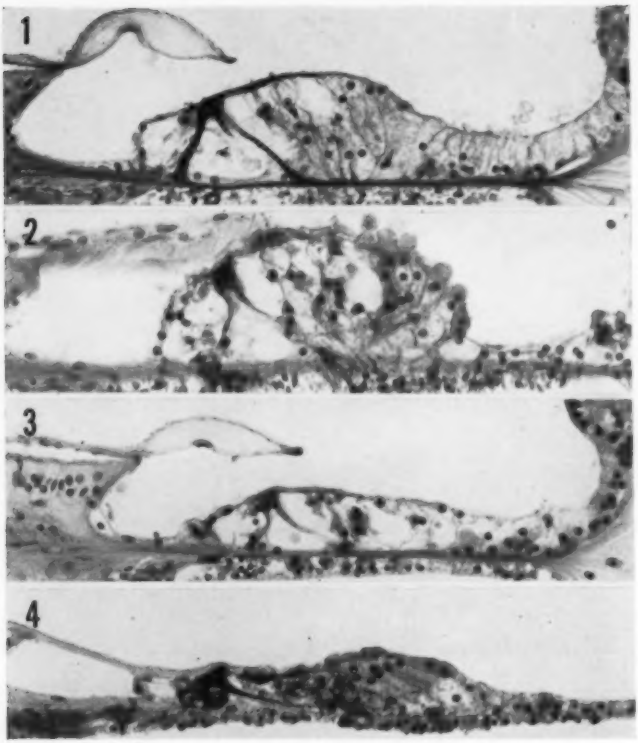


Fig. 13. Preservation of the Organ of Corti after early fixation by injecting formalin into the middle ear soon after death. 1. Good; there is edema in internal sulcus cells, and precipitates. 2. Fair; notice collapse of Reissner's membrane over Organ of Corti without compression. 3. Poor; edema of cells and intercellular spaces which give a picture of degenerative changes; however, they are incompatible with auditory function which was within normal range. 4. Unsatisfactory; compression of the Organ of Corti. 1 and 4 belong to the same subject, and the audiograms, taken 12 days before death, gave the same high tone hearing loss on both sides. Consequently compression shown in 4 was considered an artifact.

procedures are comparable; two, results were as good when injection time was 70 to 120 minutes as when it was 15 to 20 minutes; three, the ganglion of Scarpa, which can not be reached by fixative injected in the middle ear, was as well preserved in this group as it was in the group fixed by immersion only.

Postmortem changes, swelling and homogenization of cells, precipitates and globular formation, were as described in immersion procedure.

Nine of 11 cases of unsatisfactory results were caused by the compression phenomenon. The features were as previously described. This observation is in apparent disagreement with the theory that compression is an artifact, because, if early fixation was accomplished by injection, then the inner

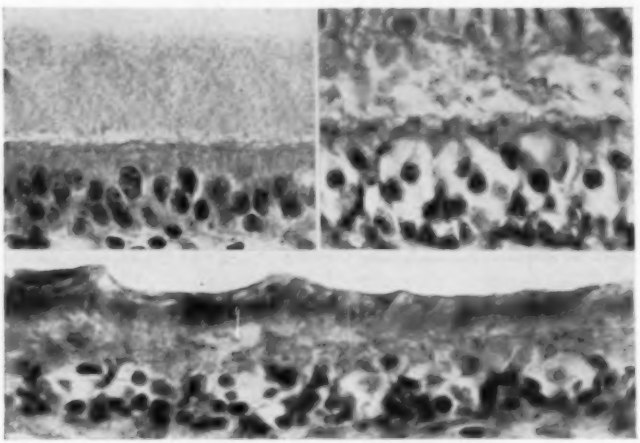


Fig. 14. Preservation of macula saccularis after early fixation by injecting formalin into the middle ear soon after death. Top left, good; right, fair; bottom, unsatisfactory result. The latter is due to compression in which the epithelium appears flattened, the cells pyknotic and otoconia agglutinated over the macula. H-E, X625.

ear structures should not be affected by subsequent treatment. The question is whether one injection of formalin into the middle ear was enough to fix the inner ear structures. From basic principles of cytology,[17] a piece of soft tissue of no more than 3 mm. in thickness requires 16 to 24 hours to be fixed, providing it is completely surrounded by the fluid. According to this, fixation of inner ear structures by injecting a fixative into the middle ear must be incomplete; the amount of formalin was small (10 cc.), the time of fixation too short

(3 to 14 hours) and the structures not completely surrounded by the fixative (round window is the main if not the only pathway). Thus, the conditions under which subsequent treatment of temporal bones was carried out might induce the compression phenomenon.

Vestibular Receptors. The degree of postmortem changes and the effect of compression upon vestibular structures were of the same order as in immersion procedure. In Fig. 14 several stages of preservation of macula saccularis are illustrated. Notice the effect of compression; the macula was somewhat flattened, and all cells were pyknotic, while otoconia and precipitates were agglutinated over the epithelium. Occasionally cell detachment and the first stages of disintegration of the receptors were observed.

TABLE III. PRESERVATION OF INNER EAR STRUCTURES.

As given by removal of the stapes before immersion, routine immersion and early fixation by injecting formalin into the middle ear soon after death.

Procedure	No.	Good	Fair	Poor	Unsatisfactory
Stapes removed	2		2		
Immersion	25	7	2	5	11
Early fixation	26	11	3	1	11
Total	53	18	7	6	22

Neural Elements. As in immersion procedure, the elements of cell bodies in Scarpa's ganglion were differentiated, and signs of postmortem changes were minimal in most of the cases. Corti's ganglion presented the same pattern of preservation in this group as in specimens fixed by immersion procedure.

VII. FINAL OBSERVATIONS.

In Table III the results of the series are summarized. The necessity of improving on histological technique is evident, since about 40 per cent of the specimens were unsatisfactory for studies of fine details, largely because of artifacts.

Both procedures, early fixation by injecting formalin into the middle ear and routine immersion, can be improved. One

injection of formalin, or other fixatives, seems to be insufficient; because a large amount of fluid must be taken up by the tissue of middle ear, and probably some runs out through the Eustachian tube. Injections repeated at intervals may be profitable. Cadavers should be refrigerated as soon as possible after death, and the temporal bones removed immediately after the cadaver is taken out of the refrigerator.

The conditions under which subsequent fixation was actually carried out may increase the progress of postmortem changes: first, the wave front of the fixative seems to take hours to penetrate through tympanic membrane, Eustachian tube, oval and round window, and other narrow pathways before reaching the inner ear structures; second, the fixative fluids were used at room temperature (about 25.5° C), and when the specimens were immersed in it heat probably reached the inner ear ahead of the fixative. The end result was, most probably, an increase in the progress of autolysis. Consequently, subsequent fixation at low temperature (4° C) is a possibility which we are now investigating.

From the results obtained in guinea pigs[1] with postmortem perfusion, it was apparent that better fixation would be possible if this method were employed to preserve human specimens. Since cadavers can not be perfused, a technique is now being developed to perfuse the temporal bones through the internal auditory artery as soon as the specimens are removed.

SUMMARY.

The purpose of this investigation was to study the preservation of human temporal bones from postmortem changes and artifacts. A study of 53 specimens was made. Three histologic techniques were compared: removal of stapes before immersion (two ears), routine immersion (25 ears) and injection of fixative into the middle ear soon after death (26 ears). The results were classified as good, fair, poor or unsatisfactory, according to the efficiency of the technique in preserving fine cellular details.

Removal of the stapes proved to be no more advantageous

than the other techniques; furthermore, it is not suitable for middle ear studies.

Early fixation by injecting formalin into the middle ear soon after death gave a higher proportion of good results than was given by immersion procedure. Fair and poor results were mainly due to postmortem changes, while unsatisfactory results were due to artifacts.

Several degrees of swelling, homogenization and pyknosis of cells, precipitates, and globular formation were characteristic of autolytic process. Disintegration, as seen in experimental animals, was not found even in specimens immersed in fixatives as long as 20 hours after death.

Unsatisfactory results due to compression phenomenon were observed in 40 per cent of the specimens included in the series. Compression was found as frequently in early fixation by injecting formalin into the middle ear as in the immersion procedure.

Several suggestions were made to improve the current histologic technique.

REFERENCES.

1. Fernandez, C.: Postmortem Changes in Vestibular and Cochlear Receptors (guinea pig). *Arch. Otolaryngol.* In press.

2. Lindsay, J. R.: Temporal Bones for Microscopic Study. *Trans. Amer. Acad. Ophthal. and Otolaryngol.*, 60:732-734, 1956.

3. Schuknecht, H. F.: Techniques for Study of Cochlear Function and Pathology in Experimental Animals. *Arch. Otolaryngol.*, 58:377-397, 1953.

4. Wittmaack, K.: Über die Intravitale Entstehung von Kollaps-und Degenerationsvorgängen an den Sinnesendstellen des Inneren Ohres. *Arch. f. Ohren-, Nasen-Kehlk.*, 112:43-52, 1924.

5. Wittmaack, K.: Über Chiningwirkung im Gehörorgan. *Zeitschr. f. Hals-, Nasen- u. Ohrenh.*, 39:211-222, 1936.

6. Mygind, S. H.: Experimental Histological Studies on the Labyrinth. III. The Endolymphatic Compression (Wittmaack's Hypotonic Degeneration). *Acta Otolaryngol.*, 33:86-116, 1945.

7. Manasse; Lange; Wolff, H. J.; Mayer, O., and Alexander, G.: Quoted from Wittmaack[4] and Mygind.[6]

8. von Békésy, G.: Description of Some Mechanical Properties of the Organ of Corti. *Jour. Acoust. Soc. Amer.*, 25:770-785, 1953.

9. Katsuki, Y., and Covell, W. P.: The Organ of Corti by Phase Contrast Microscopy. The Laryngoscope, 63:1-17, 1953.

10. WITTMAACK, K.: Über den Tonus der Sinnesendstellen des Innenhores. *Arch. f. Ohren-, Nasen-Kehlk.*, 129:118-127, 1931.

11. SCHUKNECHT, H. F.: Presbycusis. THE LARYNGOSCOPE, 65:402-419, 1955.

12. ELDREDGE, D. H.; COVELL, W. P., and DAVIS, H.: Recovery from Acoustic Trauma in the Guinea Pig. THE LARYNGOSCOPE, 67:66-84, 1957.

13. HALLPIKE, C. S., and CAIRNS, H.: Observations on the Pathology of Ménière's Syndrome. *Jour. Laryngol. and Otol.*, 53:625-655, 1938.

14. DAY, K. M., and LINDSAY, J. R.: Hydrops of the Labyrinth. Case Report. Coagulation Operation, Clinical Course and Histopathology. THE LARYNGOSCOPE, 59:213-228, 1949.

15. BUNCH, C. C., and WOLFF, D.: The Absence of Organ of Corti; an Audiometric and Histologic Study. *Ann. Otol., Rhinol. and Laryngol.*, 44:754-771, 1935.

16. OZAKI, T.: Prevention of Adverse Effects of Streptomycin on the Ear. *Arch. Otolaryngol.*, 66:673-678, 1957.

17. BAKER, J. R.: "Cytological Technique." Methuen and Co., Ltd., London, 1955.

ALUMNI ASSOCIATION OF THE NEW YORK EYE AND EAR INFIRMARY.

The Annual Spring Meeting of the Alumni Association of the New York Eye and Ear Infirmary last April was so well received that it has been decided to expand next year's meeting, which will take place from April 20-23, 1959.

Symposia and lectures on Hearing Rehabilitation, Endoscopy, and Ear Surgery will be conducted. It is also planned to offer refresher courses in Mastoid and Fenestration Surgery and Stapes Mobilization Techniques.

More complete information regarding the meeting will appear in a later issue of THE LARYNGOSCOPE.

TREATMENT OF FRACTURES OF THE FRONTAL AND ETHMOID SINUSES.*

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The traumatic disruption of the frontal and ethmoid sinuses, complicated in severe cases by dural tears, loss of brain substance and intracranial infections, presents a problem of management for both the neurosurgeon and the otolaryngologist. The degree of involvement of these paranasal sinuses following blunt or penetrating injuries to the forehead, varies from simple fracture lines to gruesome disintegration of the whole frontal area of the skull and its base. The success of any plan of treatment can best be assessed in terms of permanent recovery, and the prevention of complications early or late, such as cerebrospinal rhinorrhoea, intracranial aerocele, meningitis or brain abscess.

PURPOSE.

The purpose of this study is to review the basic principles of the neurosurgical treatment of head injuries associated with fractures of the paranasal sinuses, and to consider the aspects of the problem which are of particular interest to the otolaryngologist. The closest collaboration "between those who work below and those who work above the cribriform plate" (Cairns), will best decide the ultimate fate of the patients treated for these injuries.

INCIDENCE.

Lewin, in 1954 reported that of 1000 consecutive cases of non-missile head injuries severe enough to be admitted to the hospital, 72 (7.2 per cent) were found by radiological examination, or at autopsy, to have a fracture of the paranasal

*Presented as Candidate's Thesis to the American Laryngological, Rhinological and Otological Society, Inc., 1957.

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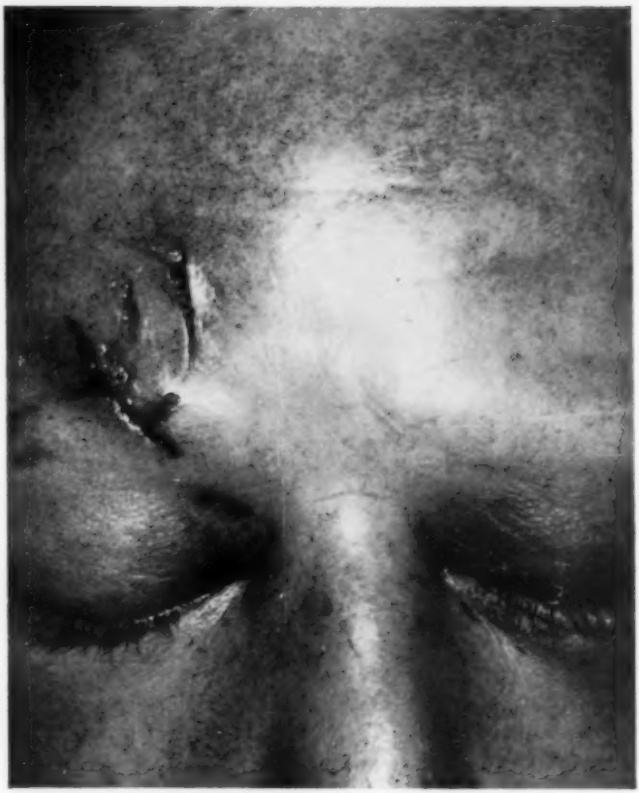

Fig. 1. The superficial appearance of the forehead gives no indication of the extent or severity of the deeper damage—Case 3.

sinuses. It is significant that approximately 20 per cent of the 72 cases with severe frontal injuries died within 12 hours of the injury, and at autopsy the majority revealed dural tears in relation to the paranasal sinuses. It is important to note, in less severe head injuries, that dural defects may occur into the paranasal sinuses with or without evidence of displacement of bone fragments, and that such defects constitute a danger which, in the absence of operative inter-

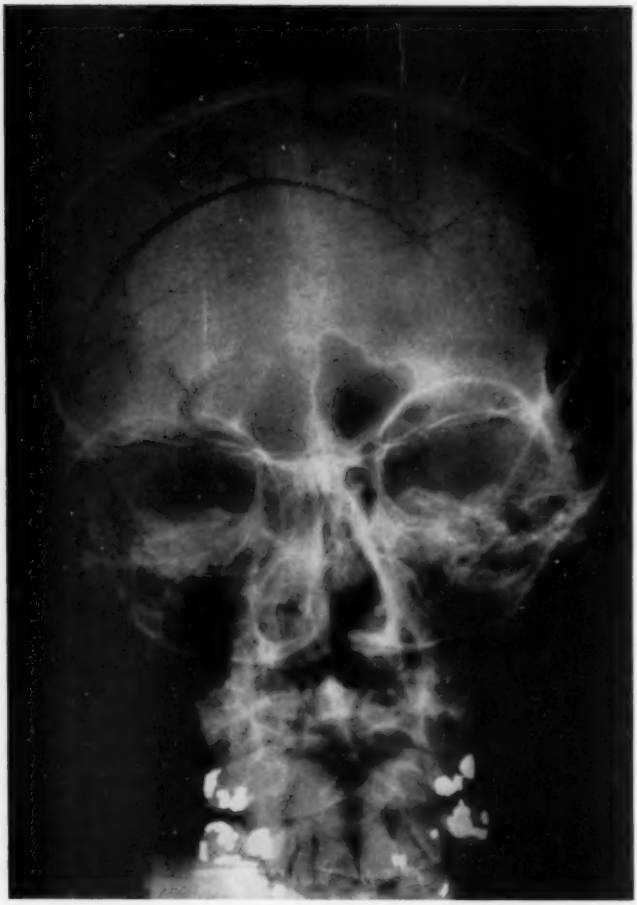

Fig. 2. Multiple compound comminuted fractures of the skull with involvement of the paranasal sinuses—Case 3.

ference, persist for years after the injury, if not indefinitely (Johnson and Dutt). In all head injuries, and in particular fronto-orbital fractures Cone, Schorstein and others have emphasized that the extent and severity of the deep damage is often considerably in excess of that which the external appearance would suggest (see Figs. 1, 2, 3).

All of these cases, therefore, demand the most careful and thorough physical and radiological examinations. The oto-

Fig. 3. The severely fractured frontal bone area seen at operation. Bifrontal scalp flap turned downwards toward the face—Case 3.

laryngologist can contribute valuable clinical information by meticulous examination of the ears, nasal passages, turbinates, meatuses and nasopharynx, for sources of cerebrospinal fluid loss or evidence of infection, and his surgical experience and skill can be utilized in the proper treatment of the traumatized sinuses in cooperation with the neurosurgeon.

THE MECHANISM OF FRONTAL AND ETHMOID SINUS FRACTURES.

The type of violence that is especially likely to produce fractures of the frontal and ethmoid sinuses is the head-on

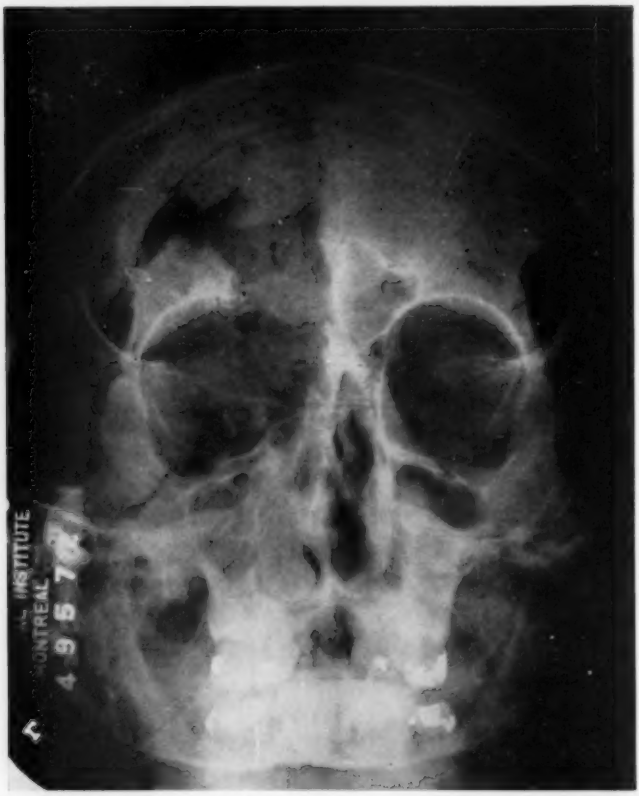

Fig. 4. The X-ray appearance of the skull following operation with ablation of the right frontal sinus and exenteration of the right ethmoid sinus—Case 3.

blow (Cairns) or blunt injury, in contrast to penetrating wounds. The ethmoid sinuses may also be involved in cases where the force of the blow is applied mainly to the facial bones.

The factors that determine the shape of the fracture must here, as elsewhere in the skull, include the size and shape of the object that inflicts the damage and the force with which

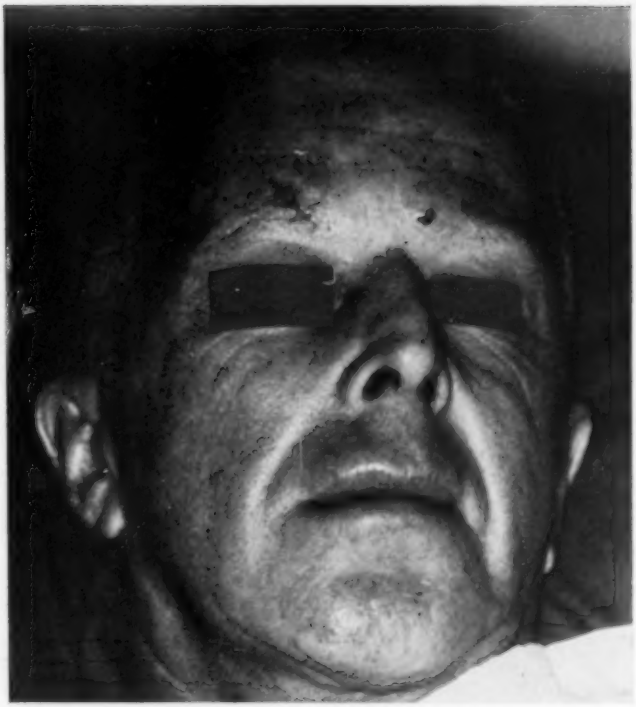

Fig. 5. The appearance of the patient after treatment—Case 3.

the skull is struck. The extent to which the frontal bone is pneumatized must also be an important mechanical determinant (Schorstein).

Fractures involving the frontal and ethmoid sinuses may occur as the result of one or more of the following mechanisms (Schorstein):

1. A force which impinges on the supraorbital arches will, if severe enough, communicate itself to the floor of the anterior fossa and tend to shorten the orbital plate in an anteroposterior direction. Should its elasticity be exceeded the bone of the anterior fossa will bend upwards and break, following

its natural curvature. In this type of direct injury or head-on blow, the dura is lacerated by displaced bone fragments, and defects are found both in the frontal sinuses and the ethmoids, frequently with separation, displacement or complete disappearance of the crista galli (Johnson and Dutt).

2. Fissured fractures may run either in an antero-posterior direction, down across the posterior wall of the frontal sinus and orbital roof or traverse the orbital plate from side to side. They are basal bursting fractures, the result of forces striking the vault of the skull above the level of the base. Their direction depends upon the direction of the injury and force, and not uncommonly they may traverse the lamina cribrosa and run into a defect in the ethmoids with an overlying dural tear.

A fissured fracture will be deflected from a direction determined by the forces which produce it, by irregularities in the thickness of the bone, and will tend to run around and isolate hillocks or blisters of thicker bone (Johnson and Dutt). Fissure fractures tend to run into the nearby foramina in the roof of the cribriform plate and ethmoid.

3. A third mechanism may act in conjunction with the two just mentioned: the depressed anterior wall of the frontal sinus, by impinging on the brittle posterior wall, may break it over a wide area, including a portion of the anterior fossa floor.

TYPES OF FRACTURES AND COMPLICATIONS.

In a study of fractures of the skull involving the frontal sinus, Teachenor (1927) was impressed with "the amazing frequency of intracranial complications from this source, and likewise the pleasing success of prompt surgical drainage in the prevention of these complications." Calvert (1942) in discussing injuries of the frontal and ethmoidal sinuses stated that "fractures of the air sinuses are more often productive of grave complications than the literature on the subject would lead one to expect," Calvert emphasized that "the chief hazard in cases of frontal or ethmoidal sinus

fracture is laceration of the dural barrier and spread of infection intracranially from the nose."

FRACTURES OF THE FRONTAL SINUSES MAY BE:

1. Simple fissured or comminuted fractures of the anterior wall of the sinus with or without displacement of bone frag-

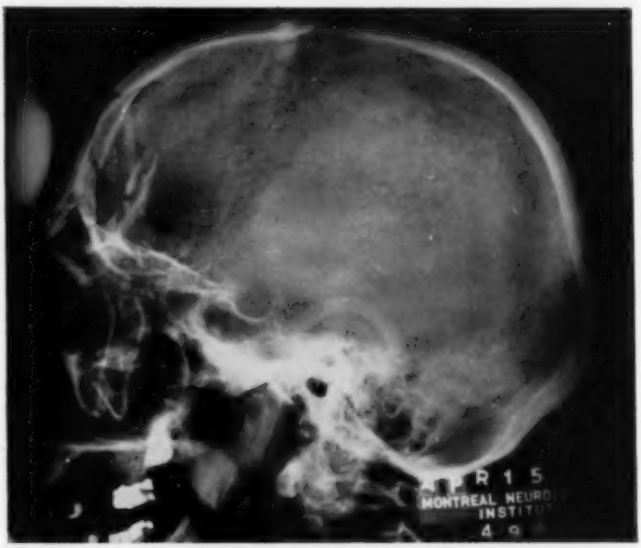

Fig. 6. Compound comminuted fractures of the frontal bone with involvement of the anterior and posterior wall of the frontal sinus.

ments. Fissure fractures may involve the posterior wall without involvement of the anterior wall (Teachenor).

2. Compound comminuted fractures of the anterior wall and floor of the frontal sinus with displacement of bone fragments into the sinus (see Fig. 6).

3. Severe compound, comminuted fractures with gross bony displacement, dural tears and pulping of brain tissue.

Cushing (1927) pointed out that mild frontal injuries may,

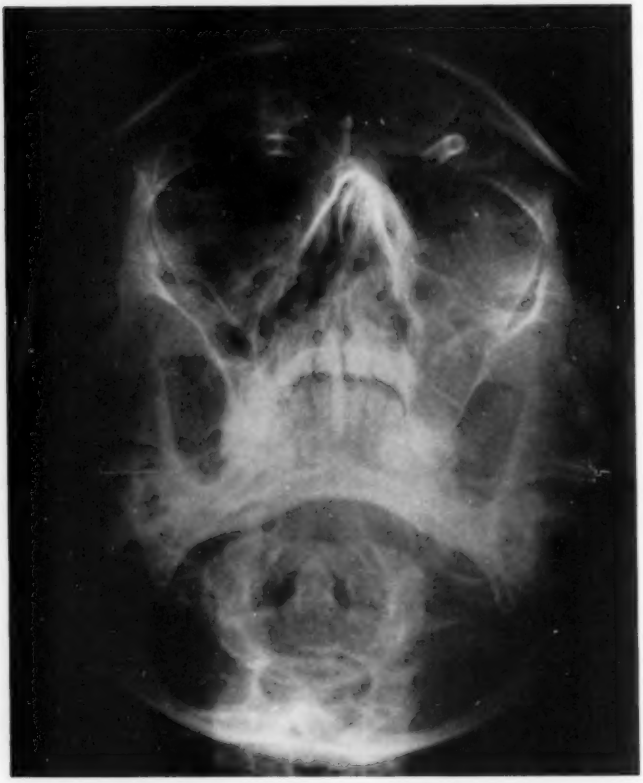

Fig. 7. Radiological appearance of the skull with rubber tubes through the trephine openings into both frontal sinuses—Case 1.

more often than suspected, produce a fissured fracture of the floor of the anterior fossa, or if not a fracture, a diastasis of the suture between the orbital plates of the frontal bone and the adjacent edge of the ethmoid bone roofing the ethmoid plates.

In the floor of the anterior fossa, as in the depressed fractures of the vault of the skull, the dural tear may not always correspond to the fracture line, but will be determined

by the sites and degree of fixation of the dura and its relative thickness. As it tears, the elastic dura slides over the cortex, and a fold is occasionally entrapped in the fracture line (Johnson and Dutt). The thin adherent dura may be penetrated by displaced spicules of bone or split by distortion accompanying a fissured fracture (McKenzie).

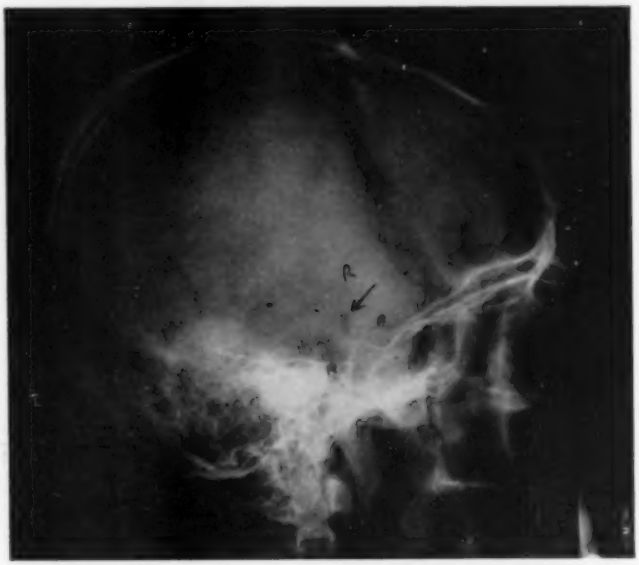

Fig. 8. Multiple fractures of the vertex and base of skull, with involvement of all the sinuses on the right side, with subarachnoid and sub-dural air—Case 2.

It is extremely rare for a fissure fracture to cross the ethmoidal roof without producing a defect in the bone and a tear in the dura (Johnson and Dutt). Because the inner periosteum of the cribriform plate is the thinnest part of the dura and, moreover, firmly anchored by its extensions through the bone along the olfactory nerves, Cairns believes that the most dangerous lesion is a fracture of the cribriform plate. In such cases there is usually an escape of cerebrospinal fluid

from the nose, anosmia may occur, and there is the obvious risk of intracranial infection.

In fractures of the frontal sinuses alone the risk of cerebrospinal rhinorrhea and intracranial infection is, in the opinion of Cairns, not so great. The dura strips more easily from the bone in this region and, even if it is torn, there is less

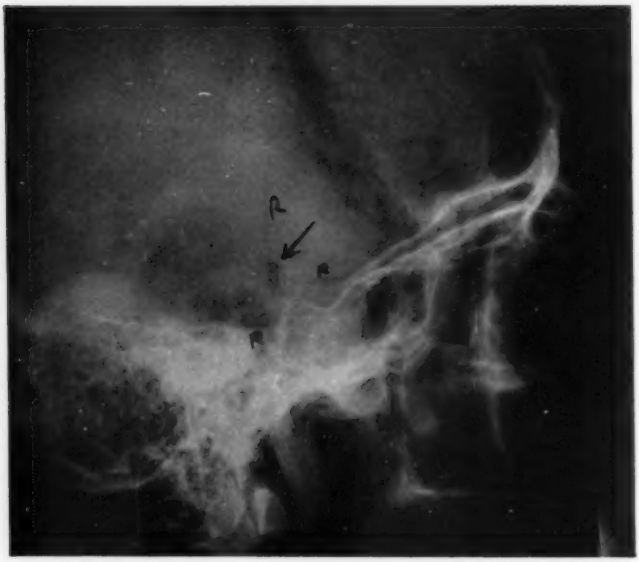

Fig. 9. Detail of Fig. 7 showing aeroceles and fissured fractures of floor of anterior fossa—Case 2.

likelihood that a cerebrospinal fluid fistula will immediately result, because the subarachnoid spaces on the frontal pole of the brain are not so large as on the inferior surface of the frontal lobe. The possibility is, therefore, greater that over the frontal pole the defect will be plugged by brain tissue (Cairns).

These opinions are in agreement with Lewis (1928), who stated that dural defects into the frontal sinus, if slight,

tended to open the subdural space; but if extensive, communicated with injured brain. Fractures involving the ethmoid region are more likely to open into the large cerebrospinal fluid lakes in the basal subarachnoid space. Lewis believed, therefore, that the risk in the frontal sinus fracture was largely one of abscess or aerocele formation, while in the ethmoidal group the risk was more one of meningitis. (Johnson and Dutt, Cairns and McKenzie).

The two unequivocal signs of dural rupture are leakage of cerebrospinal fluid from the nose and the presence of an intracranial aerocele (Calvert). Cerebrospinal rhinorrhea may follow any type of fracture involving the paranasal sinuses varying from the finest crack, just visible on X-ray, to extensive comminution and depression of the whole frontal area; but not all paranasal sinus fractures are associated with cerebrospinal fluid rhinorrhea, even when the overlying dura is torn (Lewin).

Cerebrospinal rhinorrhea may occur in the acute stage, or as a delayed complication of a head injury. Streams of clear fluid may be seen coming from the middle or superior meatuses in the nose, or on the lateral or posterior wall of the nasopharynx, with the aid of the nasopharyngoscope. A sufficient quantity can usually be collected for analysis by proper positioning of the patient's head, so that the fluid flows or drips from the nose. Cerebrospinal fluid can be recognized by its clear colorless appearance, salty taste, and chemically differentiated from thin nasal secretions by the presence of sugar and the absence of mucin. This type of complication occurred in approximately two per cent of the 1,000 head injury cases reported by Lewin (1954).

Intracranial air and cerebrospinal rhinorrhea are frequently, but not always, associated complications. Air may enter the intracranial cavity through traumatized sinuses and produce an aerocele following sneezing, straining or blowing the nose. Aeroceles may be single or multiple, subdural, subarachnoid, ventricular or intracerebral, and may occur as early or late complications.

Anosmia occurred in approximately five per cent of Lewin's

review of 1,000 cases of head injuries. It was a symptom in 38 per cent of the cases with associated sinus fractures and in 78 per cent of the cases with cerebrospinal rhinorrhea.

The extent of deep damage to the brain substance of the frontal lobes and its relationship to the dural tears, as seen at operation, varies considerably. Where there is bone missing as a result of the fracture, the brain may herniate down into the defect and later become firmly adherent to the bony margins. This is seen most frequently in the ethmoid region (Lewin). The fracture line may be enlarged by a gradual erosion of the thin margins of bone by the pulsating brain, similar to the changes in bone in contact with an aneurysm. In this type of injury the risks of epilepsy as a late complication are of considerable importance.

The presence of a nasal-intracranial connection through traumatized sinuses permits an easy pathway to the brain and meninges for infection. Meningitis may be an early or late complication, and brain abscess is more frequently a late complication, especially in cases where the fractured sinuses were inadequately treated at the time of injury.

GENERAL PRINCIPLES OF TREATMENT.

Injuries to the frontal and ethmoid sinuses frequently come within the domain of both the neurosurgeon and the otolaryngologist, "and treatment without appreciation of this fact is not conducive to the best results" (Coleman). The use of sulphonamides and penicillin has lessened the risk of intracranial infection during the acute stages after injury, and it has also effectively lowered the incidence of post-operative infection in these cases (Lewin, 1954).

The basic principles of the neurosurgical treatment of contaminated compound fractures of the vertex of the skull, with laceration of the dura and of the brain, are now standardized and in general use. The variations are those of method and timing of operative interference. Cone emphasized that "fronto-orbital wounds differ not only from wounds elsewhere in the body, but also from wounds of the vertex

with brain injury, because of the intracranial-intranasal connections through the damaged paranasal sinuses."

The basic principles of the rhinological treatment of severe compound comminuted fractures of the frontal and ethmoid sinuses are well established, but the treatment of the simple or apparently less serious fractures of these structures is not so well standardized. The treatment of the various types of fronto-ethmoidal fractures will be considered as follows:

1. Simple undisplaced fractures of this area may require no treatment (Work, 1954). It has been pointed out, however, that fissure fractures of the posterior wall of the frontal sinus may be associated with dural defects, and that such defects constitute a danger of intracranial infection that persists for years after the injury. Although the treatment of simple fractures of the frontal sinuses may be conservative if no infection is present, fractures involving chronically infected sinuses demand prompt and appropriate action to prevent serious complications. Cairns stated, however, that "the infrequency of overt sinusitis after injury suggests that intracranial infection can occur, indeed usually does occur through sinuses that are not inflamed" see Calvert, 1942).

Schorstein (1944) commented: "at the present time a certain amount of controversy exists as to the need for radical operation in those frontal sinuses where the posterior wall has been broken without a laceration of the overlying skin." He added that some advocate immediate and radical operation, and others a watching policy.

Teachenor (1927) advocated prompt surgical drainage of every case of fractured frontal sinus during the acute stage, whether there was leakage of cerebrospinal fluid or not, because "air and infection forced into the frontal sinus from the nasal cavity during sneezing or straining are important factors in the production of complications." In fissured or undisplaced comminuted fractures of the posterior wall of the frontal sinus, a simple trephine opening can be made, with a bur in the under surface of the medial end of the superior orbital ridge, through a small incision just below the eyebrow. This permits inspection of the contents of the

sinus and of the appearance of the mucous membrane. A soft rubber catheter or polyethelene tube can be sewn into place, thus permitting daily gentle irrigation of the sinus with bacitracin or neomycin in solution. The purpose of such a trephine is to prevent infection, or air, passing through the sinus into the intracranial cavity. This method of frontal sinus decompression has been advocated by Cone for many years, and it has been used in selected cases of less severe fractures of the frontal sinus with success (see Cases 1 and 2). The drainage tube is left in place until the patency of the nasofrontal duct is re-esablished and the instilled solution passes freely into the nose.

2. In compound comminuted fractures of the anterior wall and floor of the frontal sinus, the initial debridement may consist of removing bone fragments, providing closure of the skin can be accomplished and the patency of the nasofrontal duct can be established and maintained. Patency can be accomplished satisfactorily by an external ethmoidectomy and the use of an acrylic dilator (Work, 1954).

3. If the injury is more extensive and there is comminution and compounding of both the anterior and posterior walls of the frontal sinuses and fractures of the ethmoids, ablation of the frontals and exenteration of one or both the ethmoids is the procedure of choice (Work). If there are dural lacerations and evidence of loss of brain substance the surgical treatment should be carried out completely and finally at the first operation, as early as possible after the injury, by the neurosurgeon and otolaryngologist. This concept of primary treatment was strongly advocated by Cone, Stewart and Botterell.

In severe compound, comminuted fractures of the fronto-orbital region, with shattering of the paranasal sinuses, tearing of the dura and laceration, contusion and pulping of the brain, Cone removes all of the frontal bone which contains frontal sinus. The orbital plates are taken away, the glabella removed, and the orbital fascia is exposed back to the optic foramen and lesser wing of the sphenoid. The removal of the ethmoid from above is almost complete, including the per-

pendicular plates, lamina cribrosa, the crista galli and the os planum. The middle and superior turbinated processes come away with the inner wall of the lateral mass. The removal is carried back to the sphenoid region. The dura is thus exposed widely, the opening in it can be enlarged or new incisions can be made to permit debridement of the damaged

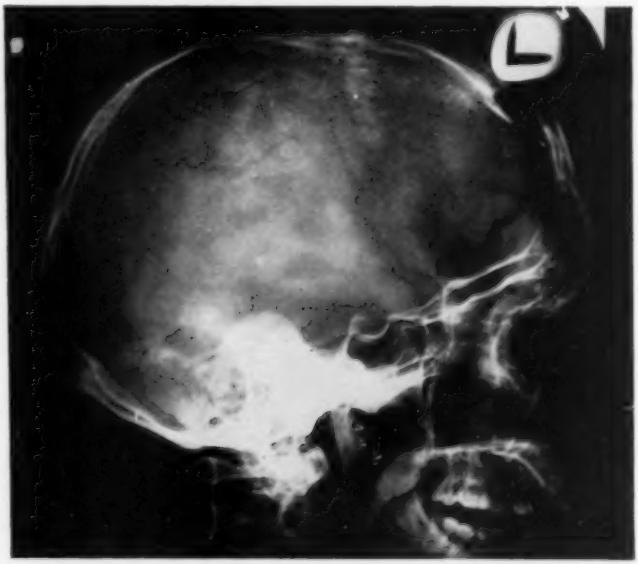

Fig. 10. Note the very severe destruction of the bones of the anterior part of the skull and disorganization of the frontal and ethmoid sinuses—Case 5.

brain. The new vault of the nasopharynx is the dura and at the sides the orbital fascia. The completely free drainage into the nasopharynx and the protection of the dura by packing until all defects are healed, are important factors in this overall method of treatment (Cone) (see Figs. 10, 11, 12, 13).

A series of five cases of head injury with fracturing of the frontal and ethmoid sinuses, and in some cases all of the sinuses, is herewith presented with comments on the type and success of treatment employed.

Case 1. Miss L. L., age 22. This patient was admitted to Montreal Neurological Institute June 20, 1942, three hours after she had fallen from her bicycle and struck her head against a cement abutment. She suffered momentary unconsciousness, a laceration 4 cm. long above the lateral half of the left eyebrow, abrasions about the left eye, face and side of the neck. There was bleeding from the nose but no definite cerebrospinal fluid rhinorrhea.

X-rays revealed fractures of the left radius and ulna, and multiple fractures of the left zygoma, left antrum, ethmoid, frontal and possibly

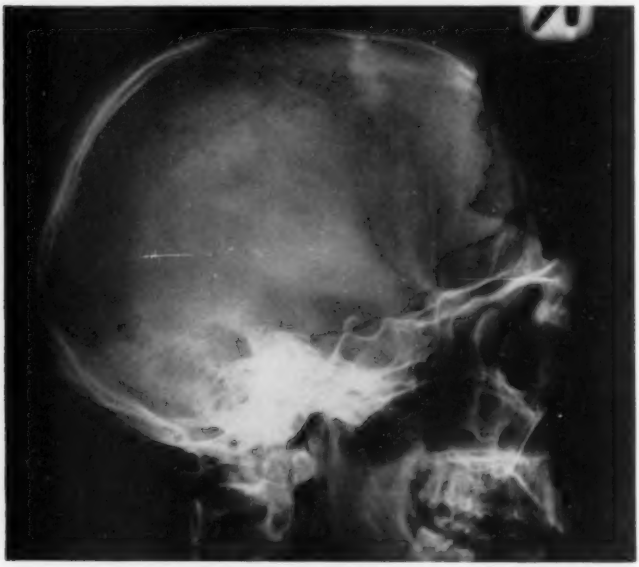

Fig. 11. After surgical removal of the grossly fragmented anterior wall of the skull—Case 5.

the sphenoid sinuses, with no bony displacement in any of these regions. There was a fracture involving the lateral portion of the anterior wall of the left frontal sinus, and there was possible involvement of the posterior wall with no displacement. Linear fissure fractures were seen in the floor and roof of the left orbit and in the lateral wall of the left antrum. All of the paranasal sinuses on the left side and the right frontal and ethmoid sinuses were cloudy.

Neurological Examination revealed a cerebral concussion with no other evidence of damage to the central nervous system.

E.N.T. Consultation June 21st. No significant findings, but in view of the X-ray findings a trephine of both frontal sinuses was recommended to prevent, if possible, serious intracranial complications.

Operation, June 22, 1942. Local anesthesia, nupercaine 1:1500. Trephine of frontal sinuses via an incision, one-half inch long, in the medial end of the eyebrow. A Cushing hand operated trephine bur was used to open into each frontal sinus. The mucous membrane in the right frontal sinus was thickened, pale and polypoidal. The nasofrontal duct was blocked by one of the polyps hanging down into its orifice. This was carefully removed, and the remainder of the mucosa was left untouched. A firm rubber tube was inserted through the trephine opening into the sinus and held in place with dermalon sutures. The lining in the left side was only slightly thickened but was ecchymotic in appearance. The

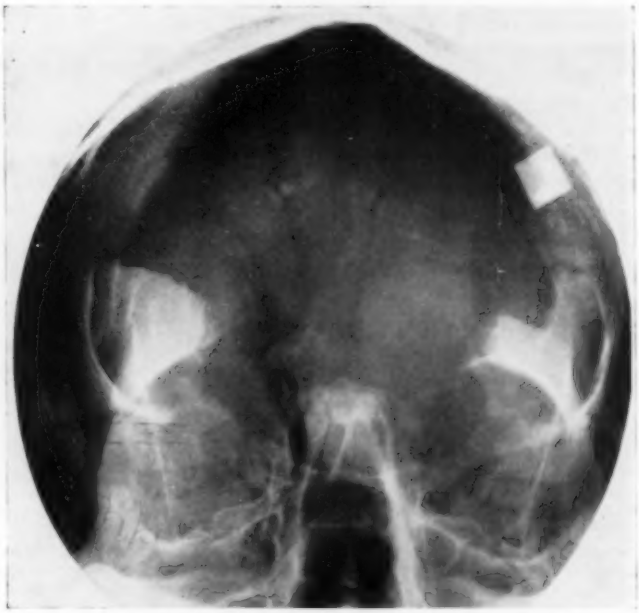

Fig. 12. X-rays showing complete removal of the frontal and ethmoid sinuses (Cone's Method)—Case 5.

cavity was filled with blood clot and serous fluid but no definite cerebrospinal fluid. A firm rubber drain was inserted as in the right side (see Fig. 6).

Progress. The dressings were changed daily and the frontal sinuses irrigated very gently with triple sulpha suspension. On the fourth post-operative day the irrigating fluid passed freely into the nose. The drains were removed on the sixth post-operative day. Sulphadiazine was given by mouth until July 4. Patient discharged on July 14.

X-rays taken on that date showed marked clearing of the sinuses.

Films taken in 1951 showed no sinus involvement, and the fractures had healed. No complications have occurred since that time.

COMMENT.

This case represents a multiple uncomplicated linear fracturing of the frontal, ethmoid, maxillary and possibly sphenoid sinuses, treated conservatively with a trephine of both frontal sinuses to prevent possible complications. The naso-

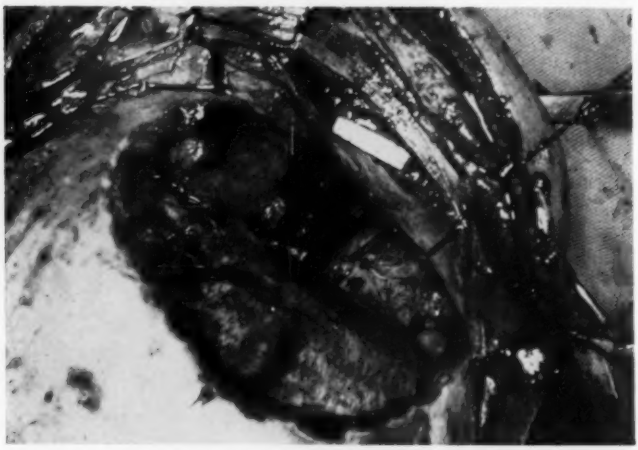

Fig. 13. Seen from above. All of the frontal bone which contains frontal sinus and compound comminuted bone fragments have been removed. Likewise the orbital plates and ethmoid sinuses completely exenterated. The dura of the frontal lobe is exposed widely. The new vault of the nasopharynx is the dura above, and the orbital fascia at the sides.

frontal ducts were apparently blocked until the fourth postoperative day. There were no intracranial complications early or late (15 years).

Case 2. Mr. G. B., age 29. This patient apparently fell 70 feet from a tower at 9:00 a.m. on March 19, 1957, and was knocked unconscious. He was flown some 1200 miles to Montreal Neurological Institute the same day. There was marked swelling and ecchymosis of the eyelids, temporal, zygomatic and maxillary areas on the right side, with a superficial brush burn over the right malar bone. He was conscious but irritable and in pain. There was no history of bleeding from either ear, but he had vomited a large quantity of blood, presumably from the back of the nose and paranasal sinuses.

Neurological Examination. Left abdominal and cremasteric reflexes absent. Right plantar upgoing to Gordon's stimulation; no involvement of the cranial nerves.

X-rays, Skull and Sinuses. There were multiple linear fractures of the lateral wall and floor of the right middle fossa and of the parietal and temporal bones, and one of these may involve the roof of the middle ear. There was air in the anterior fossa over the frontal lobes and possibly over the temporal and parietal lobes on the right side. The right antrum was completely dense and both frontal sinuses were cloudy (see Figs. 8, 9).

E.N.T. Consultation March 19, 1957. Some swelling of the right external canal. Right drum apparently intact, but there appeared to be blood and/or spinal fluid in the middle ear space. Nose—evidence of recent bleeding but no definite cerebrospinal fluid visible. In view of the X-ray findings a bilateral frontal trephine and an endonasal antrotomy were recommended.

Operation—Bilateral Frontal Trephine and Right Endonasal Antrotomy. General anesthesia. The mucosa of the left frontal sinus was grossly edematous and ecchymotic in appearance. A large fracture line was seen in the roof of the right orbit just posterior to the orbital edge; some blood clot in both frontals. Soft rubber catheters were inserted into the frontal sinuses and sewn in place with dermalon sutures. A large endonasal antrotomy was then performed on the right side.

Lumbar Puncture, March 22, 1957. Pandy, trace; protein, 180 mg. per cent; sugar, 66 mgm. per cent; chlorides, 733 mgm. per cent; cells, 3000 polys; 300 lymphs. Daily lumbar punctures for one week and weekly punctures until April 15th, showed gradual return to normal of protein, sugar, color and cell count.

Bacteriology. All cerebrospinal fluid cultures negative. Culture of nasal swab, staphylococcus pyogenes.

Course in Hospital. After drainage of the frontal sinuses was secured it was felt that there was no need for a radical neurosurgical procedure. The sinuses were irrigated daily with bacitracin and sulpha suspension. On the day of admission he developed a high fever, rigidity of the neck and positive Kernig and Brudzinski signs. A diagnosis of acute meningitis was made and vigorous systemic and intrathecal therapy was instituted at once. This was followed by early clinical improvement and complete return of cerebrospinal fluid findings to normal. He was discharged on April 17, 1957; on sulphadiazine 0.5 G t.i.d. for two weeks.

COMMENTS.

A case of multiple fractures of the vertex and base of the skull, with involvement of all the sinuses on the right side, with sub-arachnoid and sub-dural air, and development of purulent meningitis. The frontal sinuses were gently irrigated daily until satisfactory intranasal drainage was re-established. This occurred first on the left side on the sixth post-operative day and two days later on the right side.

The intra-cranial air may have entered via the sphenoids, or via the right frontal and ethmoid sinuses.

Case 3. Mr. R. S., age 50. This patient was seriously injured in an automobile accident April 12, 1950, when his wife drove into the back of a truck parked on the highway. They both sustained serious injuries and were taken to the hospital. This patient was apparently unconscious for three hours. A piece of metal was removed from a laceration on the right side of the forehead and the wound closed. He was transferred to the Montreal Neurological Institute on April 15, 1950, for further observation and treatment.

Neurological Examination. The patient was amnesic after the accident and for a period of about three days. Speech was not affected. Cranial nerves were intact. There was a laceration over the right supra-orbital region, depression of the right supra-orbital ridge and right malar eminence; bilateral periorbital ecchymosis and marked right periorbital edema (see Fig. 1).

X-rays—Skull. Multiple compound comminuted fractures of the skull with involvement of the paranasal sinuses. Multiple transverse curvilinear fractures of the frontal bone mainly toward the right side; multiple faciomaxillary fissure fractures involving both orbits, and including the frontal, ethmoid, maxillary and probably the sphenoid sinuses, especially on the right side. The right frontal, right ethmoid and the left antrum were completely airless; there was considerable thickening of the lining of the left frontal, left ethmoid and right antrum (see Fig. 2).

E.N.T. Consultation April 15, 1950. Ear drums intact and middle ear spaces clear. Nasal septum obstructing on the left side; mucosa of the right middle meatus swollen and very moist, possible cerebrospinal fluid leak, but no streaming seen; possible cerebrospinal fluid in the nasopharynx. "Decision re exenteration of the right frontal sinus to be made after neurosurgeon had exposed the area and repaired the dural tears."

Electroencephalogram April 15, 1950. "This E.E.G. suggests the possibility of severe brain damage in the right frontal region, to a lesser extent the left frontal region, and to a considerable extent in the left anterior temporal region."

Lumbar Puncture April 15, 1950. Initial pressure, 80; fluid, bloody.

Operation April 16, 1950. Bifrontal craniectomy for debridement and repair of compound depressed skull fracture, right frontal, right ethmoid, bilateral orbital; exenteration of right frontal and ethmoid sinuses.

Operation Findings. Bifrontal scalp incision, temporal fossa to temporal fossa reflected forwards. The fracture lines were found as seen in the X-rays, but were much more extensively comminuted and depressed (see Fig. 3). Th right frontal sinus contained blood and perhaps some brain. There was one large and several small tears in the dura of the orbital surface over the right frontal lobe. Fracturing of the right anterior fossa involved the right ethmoid sinuses and the orbital roof as far posteriorly as the free edge of the lesser wing of the sphenoid. There was no subdural hematoma, but some cerebral laceration. A number of free fragments of bone from the orbital roof on the left side were comminuted and had lacerated the dura.

Gelfoam impregnated with penicillin, streptomycin, bacitracin and sulfa crystals was used to fill the spaces between bone, orbital fascia and nasal cavity; the left frontal sinus was trephined and a small rubber catheter inserted into the sinus through a stab incision in the eyebrow.

Post-operative Course—X-rays April 21st. The orbital roofs, the cribriform plate and the right lamina prapacea of the ethmoid bone were

removed, and the fragments of bone around the right frontal region adjusted until they are now in quite good position (see Figs. 4, 5).

May 23, 1950. Patient had a remarkably good recovery (five weeks). There was some periorbital swelling on the right side three weeks post-operatively associated with some infection (staphylococcus pyogenes) in and about the right sphenoid sinus and in the nose. The new roof of the nasopharynx is clean and covered with mucous membrane.

E.E.G. May 15th. Shows residual severe damage, maximum over the right anterior frontal region.

COMMENT.

This procedure was carried out as an emergency operation four days following extensive compound comminuted skull fracture. Its chief purpose was to close the dural defects and prevent intradural septic complications. This was accomplished in this case with less radical removal and less deformity than usual.

Case 4. Mr. M. R., age 26. This patient was struck by a car and knocked unconscious on August 1, 1942. He was semi-conscious when admitted to the Neurosurgical Service one hour later. He had a contused area above the root of the nose, ecchymosis with marked swelling of both eye-lids, and superficial lacerations and abrasions of the face, body and extremities. He was bleeding extensively from the nose and mouth, with vomiting of blood at times.

Neurological Examination. Cranial nerves intact; reflexes normal; cerebral concussion, fracture of base of skull and facial bones.

X-rays—Skull and Sinuses. Aug. 1, 1942: Multiple fractures involving the frontal bone, ethmoid and nasal bones and both maxillae. The fracture lines were roughly stellate in the frontal region with the point of convergence in the outer table immediately overlying the left frontal sinus with definite involvement of the frontal sinuses on both sides. The major fracture line in the left frontal bone showed no depression. There was, however, quite marked depression of a circular area involving the frontal sinuses, nasal bones and ethmoid bones. The right antrum was diffusely dense, the left antrum relatively clear; the sphenoids were clear; the lower orbital margins and the roofs of the antra were involved on both sides.

E.N.T. Consultation. Ears normal; nose, some deformity and swelling over the bridge, and both sides filled with blood and blood clots. Blood clots in the nasopharynx; throat clear except for postnasal bleeding.

Operation, August 1, 1942. Through an incision in the left and right eyebrows the anterior and posterior walls of the left frontal sinus were found to be extensively comminuted. Loose fragments were present in the posterior wall of both frontal sinuses. Both sinuses were filled with blood and clots. The mucous membrane was removed from both frontal sinuses. The fracture lines were followed back to the posterior ethmoid cells and fragments of bone were removed from this whole area. At only one point was the dura opened; this was to the left of the crista galli. Only a slight amount of cerebrospinal fluid was seen. When the debridement was completed a large rubber tube was passed from above down through the nose. The cavity left after removal of the frontal sinuses and ethmoid air cells was packed with gauze soaked in sulfona-

mide and elphamel ointment mixture; a Penrose drain was passed out through a seperate stab wound; the incision was then closed carefully.

On Aug. 14th and 24th the original incision was reopened, the cavity inspected, irrigated and re-packed as in the original operation. The hard rubber drain was removed on Aug. 14, and replaced by a Penrose drain; healthy granulations covered the exposed dura; the visible deformity over the forehead was minimal.

On Sept. 11, 1942, the patient had a generalized seizure.

Encephalogram, Sept. 12, 1942—Showed a marked general dilatation of both lateral ventricles, also the third ventricle. The appearance suggested at least partial obliteration of the subarachnoid space over the surface of the two hemispheres. The patient has had no further complications since discharge from hospital Sept. 29, 1942.

Case 5. Mr. M. C., age 23. On Nov. 25, 1949, this patient was struck on the forehead by a falling log in a lumber camp. He was admitted to hospital in an unconscious state with a compound comminuted fracture of the frontal bones and sinuses. Brain tissue was found extruding from the lacerations. At operation a frontal skin flap was turned, the dura over the left frontal was found torn and the wound filled with bone fragments. The dura was apparently not sutured, drains were inserted and the skin closed. On penicillin and streptomycin the patient did fairly well until he developed an obvious wound infection. He was transferred to the Montreal Neurological Institute on Dec. 13, 1949, in a state of stuporous confusion with a purulent discharge from the frontal laceration.

X-rays—Skull, Dec. 13, 1949. A large, approximately round fragment of bone containing the upper portions of both frontal sinuses was seen lying in the middle of a large frontal bone defect. It was bulging outwards about one cm., signifying considerable increase in intracranial pressure. There was extreme comminution of the central portion of the floors of both anterior fossa. The cribriform plate was markedly comminuted with the medial halves of the orbital plates of the frontal bone. There were multiple fractures of the laminae papyracea of the ethmoid, of the lesser wings of the sphenoid, of the inferior orbital margins, and of the left side of the skull. All of the paranasal sinuses were dense.

Electroencephalogram, Dec. 13, 1949. "These findings suggest a diffuse destructive brain process in both frontal lobes." This patient had infected frontal fractures with brain abscess and osteomyelitis.

Operation, Dec. 13, 1949. Pansinusectomy and debridement of the frontal lobe. The frontal and ethmoid sinuses were completely removed, *i.e.*, the roof and medial wall of the orbits, the supraorbital ridges, the cribriform plate and crista galli (which was floating in pus), the superior and middle turbinates. The ostea of the antra were enlarged and the mucous membrane of the antra removed. The sphenoid sinuses were entered anteriorly and the mucous membrane removed, all through this intracranial approach. The damaged and infected brain and dura was then debrided; complete dural closure was found to be impossible. The wound was irrigated with sulfa crystals and bacitracin repeatedly. The nasal cavity was closed off with gauze packs soaked in the same solution and the frontal cavity packed with gelfoam, soaked in the same solution and powdered with bacitracin; the skin was closed with 00000 nylon sutures.

Course in Hospital. Despite this extensive infection of the bone, sinuses and brain, this patient did remarkably well; his temperature was 104 on admission, and with the aid of penicillin and streptomycin was 99 in two days, and it remained down thereafter. The nasal packs

were removed in two weeks, and by the end of six weeks all of the crusts, and apparently all of the gelfoam had come away; there was no discharge from the nose after three weeks from the time of operation.

Eleven months after the accident the patient had a generalized convulsion. In the following 18 months he had eight to ten attacks, and on March 17, 1952, he was admitted to the Neurosurgical Service for treatment. Bilateral meningo-cortical scars were removed; patient discharged improved.

CONCLUSIONS.

The incidence, mechanisms and types of fractures of the frontal and ethmoid sinuses occurring in head injuries, and the various complications, have been reviewed and discussed. The basic principles of the rhinological and neurosurgical treatment of this particular group of clinical problems have been considered, and comments have been made on the cases presented to illustrate these principles. From all this data certain aspects of the problems involved, which are of particular interest to the otolaryngologist, have become apparent:

1. The frequency of complications following even simple fissured or comminuted fractures of the frontal and ethmoid sinuses demands recognition.

2. The external appearance of the injured area gives no indication of the extent nor of the severity of damage to the underlying bones, sinuses, dura and brain substance.

3. Because serious intracranial complications may occur after not only a severe but apparently a minor head injury, when associated with fractures of the paranasal sinuses, all of these cases deserve the most complete and thorough physical and radiological examinations.

4. These cases should be examined, evaluated and treated as soon as possible after the injury.

5. If there is definite evidence of dural lacerations and damage to the brain substance, it is logical that the surgical treatment should be carried out completely and finally at the first operation, by the neurosurgeon and otolaryngologist working in close cooperation. The collaboration of these two specialties will best decide the ultimate fate of the patients treated for these serious injuries.

ACKNOWLEDGMENT.

The author expresses his sincere appreciation and gratitude to Dr. W. J. Cone, Neurosurgeon-in-Chief at the Montreal Neurological Institute, for his encouragement and assistance in preparing this paper, for the use of his photographs and for his patient instruction and guidance in this type of surgery in the past 18 years.

BIBLIOGRAPHY.

CAIRNS, HUGH: Injuries of the Frontal and Ethmoidal Sinuses with Special Reference to Cerebrospinal Rhinorrhea and Aeroceles. *Jour. Laryngol. and Otol.*, 52:589-623, Sept., 1937.

CALVERT, C. A.: Discussion on Injuries of the Frontal and Ethmoidal Sinuses. *Jour. Laryngol. and Otol.*, 57:499-508, July, 1942.

COLEMAN, C. C.: Fracture of the Skull Involving the Paranasal Sinuses and Mastoids. *Jour. A.M.A.*, 109:1613-1616, Nov., 1937.

CONE, W. J.: See Calvert, Stewart and Botterell.

CONE, W .J.: Personal Communication and Unpublished Papers.

CUSHING, H.: Experiences with Orbital-Ethmoidal Osteomata Having Intracranial Complications. *Surg., Gynec. and Obstet.*, 44:721, June, 1927.

JOHNSON, R. T., and DUTT, P.: On Dural Laceration Over Paranasal and Petrous Air Sinuses. *Brit. Jour. Surg., War. Surg.*, Supp. 1, p. 141, 1947.

LEWIN, W.: Cerebrospinal Fluid Rhinorrhea in Closed Head Injuries. *Brit. Jour. Surg.*, 42:1-18, July, 1954.

LEWIS, A. J.: Traumatic Pneumocephalus. *Brain*, 51:221-243, 1928.

MCKENZIE, DONALD: Head Injuries Involving Air Sinuses. *Brit. Med. Jour.*, 1:652, May, 1944.

SCHORSTEIN, J.: Compound Fronto-Orbital Fractures. *Brit. Jour. Surg.*, 31:221-230, Jan., 1944.

STEWART, O. W., and BOTTERELL, E. H.: Cranio-Facial-Orbital Wounds Involving Paranasal Sinuses; Primary Definitive Surgical Treatment. *Brit. Jour. Surg., War. Surg.*, Supp. 1, p. 112, 1947.

TEACHENOR, F. R.: Intracranial Complications of Fracture of Skull Involving Frontal Sinus. *Jour. A.M.A.*, 88:987-989, March, 1927.

WORK, WALTER P.: Paranasal Sinuses in Relation to Skull Injury; Subsequent Defects and Their Correction with Tantalum Plates. *Jour. A.M.A.*, 141:977-981, Dec., 1949.

WORK, WALTER P.: Trauma to the Frontal Sinuses. *Arch. Otol.*, 59:54-64, Jan., 1954.

PROBLEMS IN THE DIAGNOSIS OF PHARYNGEAL PARESIS.*

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Within the narrow confines of the hypopharynx, an area comprising the pyriform sinuses and postcricoid area, one may encounter a surprisingly large variety of disorders producing symptoms. Mechanical obstruction by neoplasm or stricture may produce dysphagia, aspiration, and cough. Narrowing of the lumen by mucosal atrophy in Plummer-Vinson syndrome or encroachment on the lumen by cervical exostoses may interfere with swallowing. Pulsion diverticulum may produce dysphagia, regurgitation, and cough. Increase in the tonus in the cricopharyngeus muscle may cause vague sensations of tightness and choking.

These conditions can usually be diagnosed by a careful history and a combination of physical, neurological, radiological and endoscopic examinations. The exception to this general rule is a disorder which may present a perplexing problem in diagnosis even after thorough investigation with all available techniques; namely, isolated pharyngeal paresis. This condition is usually due to a lesion in the upper portion of the vagus nerve trunk and may be the result of injury by chemical or bacterial toxins, by trauma, tumor, certain degenerative disorders or by localized vascular accidents in the region of the nucleus ambiguus. Supranuclear lesions may also produce dysphagia and voice changes if they involve the corticobulbar tracts of both sides.[1-10]

Pharyngeal paresis may exist in the absence of other neurological disturbances or may precede them and may closely simulate neoplasm of the hypopharynx both by mirror examination and by roentgenogram. Of approximately 15 cases

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of nonpoliomyelitic pharyngeal paresis admitted to this hospital during the past three years, five were sent in with an admission diagnosis of carcinoma of the hypopharnx or upper esophagus because of various combinations of dysphagia, "exudate" in one pyriform sinus, "edema" and fullness of one side of the larynx, and other suggestive findings.

Curiously enough, the failure to find tumor by endoscopy may still leave the diagnosis in doubt, a fact dramatically illustrated during my internship, in an elderly woman admitted with dysphagia of several months' duration. Barium studies were interpreted as showing a space-occupying lesion of the hypopharynx. The "filling defect" on X-ray was so strongly suggestive of neoplasm that after esophagoscopy failed to reveal tumor, surgical exploration of the neck was carried out on the assumption that an extrinsic mass was present and was making pressure on the hypopharynx. In retrospect, it is obvious that the asymmetry in the barium-filled hypopharynx was due to inadequate emptying by the atonic musculature on one side, particularly since a diagnosis of neurological disease was made before the patient left the hospital.

There are certain remarkable similarities between pharyngeal paresis and carcinoma of the hypopharynx in the history, physical and endoscopic examination, and by radiological studies.

HISTORY.

A. Dysphagia. This may be present in either condition. In carcinoma of the hypopharynx, it results from mechanical obstruction at the esophageal inlet, tends to come on rather slowly, and is noticed first for solids, then for liquids.

In pharyngeal paresis, dysphagia results from involvement of the constrictor muscalature of both sides. Although complete unilateral paralysis has been shown to be compatible with normal deglutition,[5,10] many patients with dysphagia present a predominantly unilateral atony of the pharynx on radiological examination with barium. It is probable that both sides have been involved in the neurological disorder,

but that the weakness is greater on one side. This is particularly apt to occur in patients with a previous mild cerebral vascular accident.

B. Hoarseness. A postcricoid carcinoma, which involves the anterior wall of the hypopharynx, may infiltrate the cricoarytenoideus posticus muscle, the strong abductor of the cords. This produces a limitation of motion and resultant changes in voice.[11] A postcricoid or pyriform sinus tumor may also produce hoarseness by infiltrating laterally and inferiorly to involve the recurrent laryngeal nerve.

Vagus nerve lesions located between the nucleus ambiguus and the ganglion nodosum cause paralysis of the cord, usually in a para-median position.[5,10]

C. Cough and Choking. In carcinoma of the hypopharynx, local irritation may produce cough. In both tumor and paresis, inadequate emptying of the pharyngeal recesses allows aspiration of the excess secretions with resultant cough.

PHYSICAL FINDINGS.

A. Mirror Examination. Indirect examination of the larynx and hypopharynx may be very uninformative. Unless a tumor has outgrown the depths of the pyriform sinus or postcricoid space, there may be no view of the neoplasm itself. A motionless vocal cord may be present in either condition (see above). Saliva or food lodged in the pyriform sinus on the paralyzed side may look like exudate and may simulate a keratinizing neoplasm.

What looks like edema may actually be relaxation and bulging of the atonic musculature of the ventricular band or of the lateral pharyngeal wall. This may simulate a tumor deep in the pyriform sinus.

B. Palpation of the Trachea. Palpation of the trachea may reveal forward displacement in either condition: In postcricoid tumor by pressure from behind the cricoid, in pharyngeal paresis by relaxation of the cricopharyngeus muscle which usually supports the cricoid tightly against the cervical vertebrae.[12] What is more remarkable, the trachea may devi-

ate from the mid-line position in either condition: in neoplasm, as the result of pressure from postero-laterally; in paresis, from relaxation of the supporting musculature on one side. Deviation of the larynx and trachea in unilateral paresis of the pharynx is rather difficult to explain, yet it has been seen in two cases. Two possible mechanisms for this deviation suggest themselves: *1.* relaxation of the middle and inferior constrictors of one side, allowing those of the active side to rotate or displace the larynx and hyoid; and *2.* lateral pressure by food retained in one pyriform sinus.[13]

NEUROLOGICAL EXAMINATION.

In both pharyngeal paresis and hypopharyngeal carcinoma the patient may initially show no other sign of disease. Weakness of the other three posterior cranial nerves should be looked for, and movements of the soft palate, tongue, posterior wall of the pharynx, sternocleido mastoid and trapezius muscles observed. Certain disorders of the posterior group of cranial nerves have been described by Schmidt, Avellis, Vernet, Jackson, Tapia, and Collet-Sicard.[14] These syndromes consist of various combinations of dysphagia, hoarseness, dysarthria, nasal regurgitation, and sensory and motor disturbances of the trunk and extremities. They are usually due to obvious neurological disorders and do not present the type of problem in differential diagnosis under discussion here.

RADIOLOGICAL EXAMINATION.

This usually consists of a plain antero-posterior view; lateral view of the neck, fluoroscopic study of the barium swallow and comparison of the two sides of the barium-coated hypopharynx during the Valsalva maneuver. Careful radiological examination is probably the most important single measure in the evaluation of pharyngeal paresis of any type.[15-21] Here again, however, there are certain pitfalls:

1. Lateral Film. The plain lateral film is one of the most useful views in distinguishing between tumor and paralysis in this area, and yet there may be a soft tissue mass behind

the larynx in either condition; in tumor, because of neoplasm; in paresis, because of the anterior sagging of the atonic inferior constrictor muscle or because of spastic contraction of the muscle in supra-nuclear lesions.

2. Barium Swallow Film. "Filling defect" may be deceptive and may represent the normally emptied side of the hypopharynx after the swallow has been completed. Fluoroscopic observation during swallowing will show that the radio-opaque side of the hypopharynx is paretic.

Since certain individuals tend to use only one pyriform sinus in swallowing a small amount of fluid, the filling ability of both pyriform sinuses must be checked by: *1.* having the patient turn his head first to one side, then to the other while swallowing, so as in each case to direct the fluid into the opposite pyriform sinus; *2.* having the patient swallow a large enough amount of barium to fill both pyriform sinuses at the same time. It may be difficult to get the patient to swallow a large mouthful of fluid since he realizes that even small amounts will produce coughing. Templeton[16,17] states that a paretic pharynx will accommodate larger amounts of barium mixture if this is administered while the patient is in a supine position. In this position, both pyriform sinuses will fill initially as is shown by Fig. 7.

Food particles which may have been retained in the pyriform sinus should be looked for before barium studies are done.

3. Valsalva's Maneuver. The Valsalva maneuver, a forced expiratory effort against a closed mouth and nose, may outline the superior edge of a tumor, or it may show ballooning of the atonic side in unilateral pharyngoplegia. This is a most useful diagnostic test.

CASE HISTORIES.

Case I. Dr. E. H., 71-year-old dentist, admitted with inability to swallow of four days' duration.

Present Illness—Four nights before admission he awoke from sleep with tightness of the throat, moderate intermittent dyspnea, and hoarseness; on admission he could not swallow even fluids, and his voice was hoarse.

Mirror Examination—There is puddling of milk and saliva in the right pyriform sinus. The right vocal cord is motionless in the midline. The right false cord is markedly edematous, and there is a grayish mass visible deep in the pyriform sinus below the arytenoid cartilage on the right side.

Impression—Carcinoma of the right pyriform sinus or hypopharynx with involvement of the right recurrent laryngeal nerve.

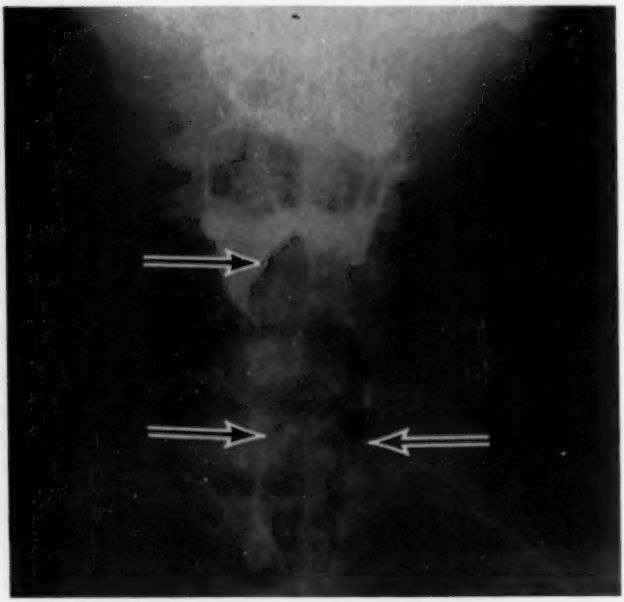

Fig. 1. (E.H.) Irregularly outlined area of radiolucency behind the cricoid cartilage (upper arrow), with puddling of barium in the hypopharynx and valleculae, chiefly in right pyriform sinus. Dilatation of right pharyngeal wall and deviation of trachea to left (lower arrow). Compare this picture of pharyngeal paresis with Fig. 3, retrocricoid carcinoma.

X-ray—There is displacement of the trachea to the left in the region of the lower cervical and upper dorsal vertebrae; no well defined mass is noted. In the region of C 5 and 6 there is a soft tissue swelling with an indentation on the posterior wall of the trachea in that area (see Figs. 1, 2).

Esophagoscopy—Swelling of the right lateral pharyngeal wall at the cricoid level. Right cord motionless, questionable bulging of the posterior tracheal wall. Esophagoscopy negative for mucosal lesion.

X-ray—(two days after admission). (Thin barium). The valleculae and pyriform sinuses bilaterally show no pathology. The contrast medium

appears to encounter an obstruction at the level of the junction of the pharynx with the esophagus beyond which no contrast medium is noted in the esophageal passage. There is a small amount of regurgitation of barium into the trachea, and this is noted in the over-exposed film in the left main bronchus.

X-ray Diagnosis—

1. Obstruction of proximal-most portion of esophagus.

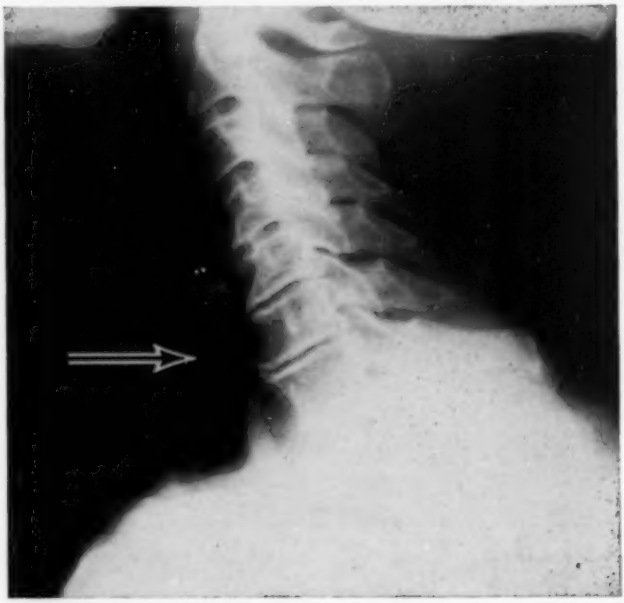

Fig. 2. (E.H.) Soft tissue density in retrocricoid area, the result of atony of the cricopharyngeus muscle. Compare with Fig. 4, retrocricoid carcinoma.

2. Soft tissue mass opposite C 6.

3. Hypertrophic osteoarthritis of the cervical vertebrae.

ENT Consultation—(Two days after admission). Mirror examination revealed right vocal cord in a para-median position; voice husky; both vocal cords inflamed and swollen; probably foreign body, which has penetrated the mucosa and is causing pari-esophageal inflammation.

Over the next three days the patient gradually improved and was able to swallow a little fluid. There was less edema noted in mirror examination of the larynx; several large grayish brown masses were coughed up by the patient.

X-rays—(Six days after admission). There is failure to opacify completely the left pyriform sinus; however, under fluoroscopy this pyriform sinus appeared to be well visualized, and not remarkable in any respect. The trachea deviates to the left in region of C 6 and 7. There is a small trickle of barium into the esophagus; no evidence of filling defect.

X-ray diagnosis—Incomplete obstruction of the proximal esophagus, probably due to a central nervous system lesion or secondary edema.

Esophagoscopy—(Eleven days after admission); no foreign body or mucosal lesion found.

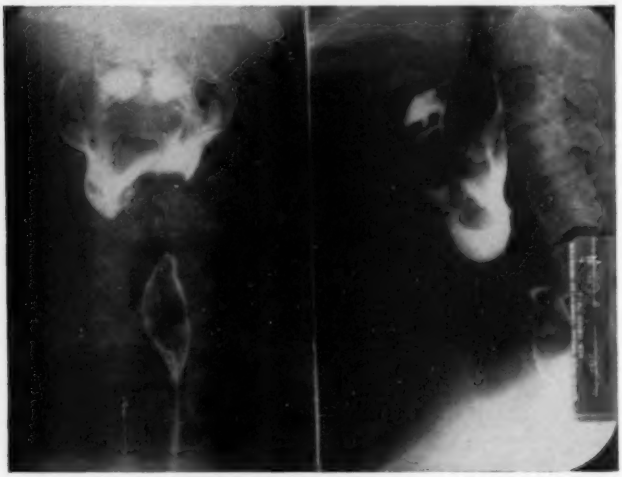

Fig. 3. (V.W.) Valvalva maneuver showing slight dilatation of pharynx, particularly of right pyriform sinus, in a case of retrocricoid carcinoma. Compare with Fig. 1.

Diagnosis—Bulbar lesion (Vascular) involving Nerves X and IX.

After three weeks the swallowing had improved. The patient could take a liquid diet, and the final impression was that there was a vascular lesion localized in a very small area of the bulb, probably an embolus. As the patient's temperature was never over 99.2° rectally; urinalysis and blood picture normal, it is unlikely that infection was a prominent etiological factor here.

Follow-up—Two years later, this patient had a cerebrovascular accident with temporary paralysis of the left side from which he recovered. When check-up was attempted five years after original admission he had moved to Florida. Voice was said by his son to be adequate, but slightly weak; he eats mostly soft food.

Discussion—The history, mirror examination, and the in-

itial radiological examination in this case suggested a neoplasm of the upper esophagus or hypopharynx. The trachea deviated away from the side of the paralysis and was due in this case, not to pressure from a tumor situated in the hypopharynx, but probably to relaxation of the constrictor musculature attached to the right side of the larynx. The "edema"

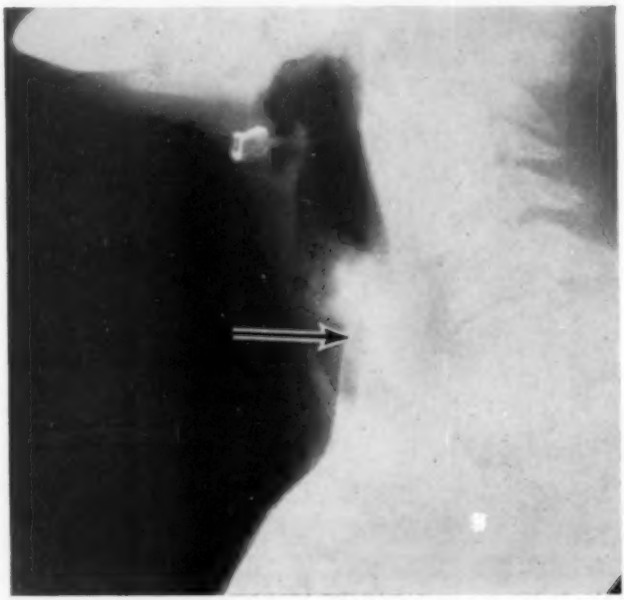

Fig. 4. (V.W.) Retrocricoid soft tissue density, with some forward displacement of posterior wall of trachea. Compare with Fig. 2, postcricoid carcinoma.

of the larynx seen on mirror and direct endoscopic examination probably represented relaxation and bulging of the atonic inferior constrictor muscle, and accounted for the soft tissue swelling behind the larynx on the lateral X-ray view. The grayish mass in the pyriform sinus was identified in this case as retained food (junket), in others as saliva and epithelial debris from the mouth.

Case II. R. C., 68-year-old woman, admitted with chief complaint of hoarseness and dysphagia, six months.

Present Illness—Six months ago the patient developed hoarseness over a period of a few days. Shortly thereafter she developed progressive dysphagia so that she can now take only soft foods and liquids. She has lost about 30 pounds over this period of time; has occasional regurgitation of food or liquid into the mouth; has a morning cough productive of yellow sputum.

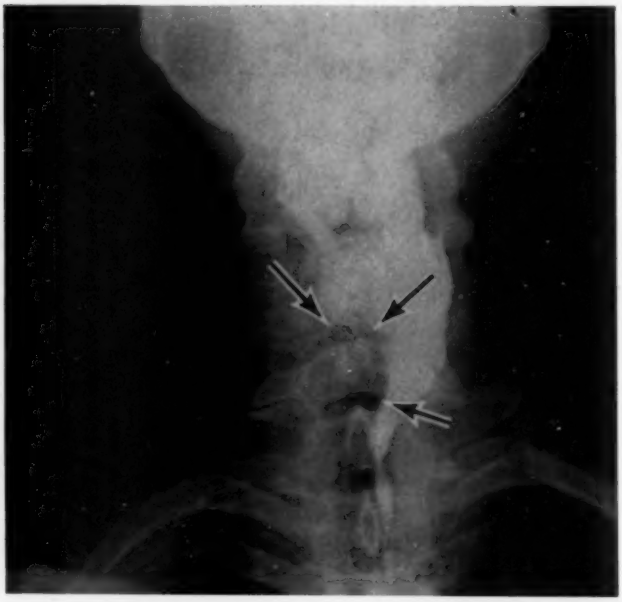

Fig. 5. (R.C.) Irregularly outlined soft-tissue density, with retention of barium in valleculae and left pyriform sinus. The soft-tissue density in the right pyriform sinus area is illusory, and represents the normal hypopharynx after completion of swallow. The opposite side, both hypopharynx and larynx are paralyzed.

Past History—Seven years ago, left mastectomy for a carcinoma without lymph nodes. One year ago right mastectomy, intraductal proliferation without malignancy.

Mirror Examination—Left cord is motionless and lies in paramedian position; patient is hoarse. There is a collection of saliva in the left pyriform sinus.

Physical Examination—Blood pressure 175/110. There is a 0.5 cm. nodule under the intact skin on the left in the scar of the mastectomy; it is firm and movable. Laboratory work showed no abnormalities of

the blood or urine; Wassermann negative. There are no cervical or supra-clavicular lymph nodes palpable. The left shoulder droops noticeably and is limited in motion.

X-ray—Metastatic involvement of dorsal spine, L 3 and 4; left sacrum; left ileum, and left ischium. Minimal fibrotic tuberculosis, right apex. Bilateral apical plaural thickening. Poor filling of the right pyriform sinus; no displacement of the barium-filled esophagus.

Diagnosis—Metastatic lesion involving the right pyriform sinus (see Fig. 5).

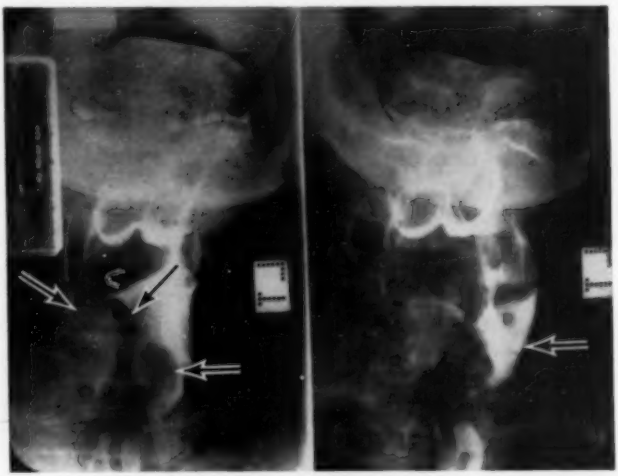

Fig. 6. (H.R.) Film on left demonstrates the pattern which was originally interpreted as soft-tissue mass in right hypopharynx. Valsalva maneuver on right half of plate shows dilatation of left pyriform sinus, and strongly suggests paresis as the underlying disorder.

Endoscopy—No evidence of tumor in the hypopharynx or the upper esophagus. The lumen was patent and admitted an esophagoscope; left vocal cord motionless.

Impression—Bulbar lesion in region of nucleus ambiguus. Absence of upper mediastinal mass on X-ray with signs of Nerve X and XII involvement suggests central rather than peripheral lesion.

Discussion—The X-rays in this case show a failure of the right pyriform sinus to fill with barium. Absence of tumor in this area on endoscopic examination indicates that the right hypopharynx is the normal side and has emptied itself normally (see Fig. 5). The puddled saliva and residual barium in

the left pyriform sinus is the result of atony of the pharyngeal musculature on that side. Fluoroscopy and the Valsalva maneuver after barium swallow demonstrate this vividly.

Case III. H. R., 68-year-old man, admitted with three-week history of hoarseness and dysphagia. Pre-admission roentgenograms had been interpreted as showing a "tumor of the hypopharynx" (see Fig. 6).

Endoscopy—The right vocal cord is slightly sluggish. There is saliva

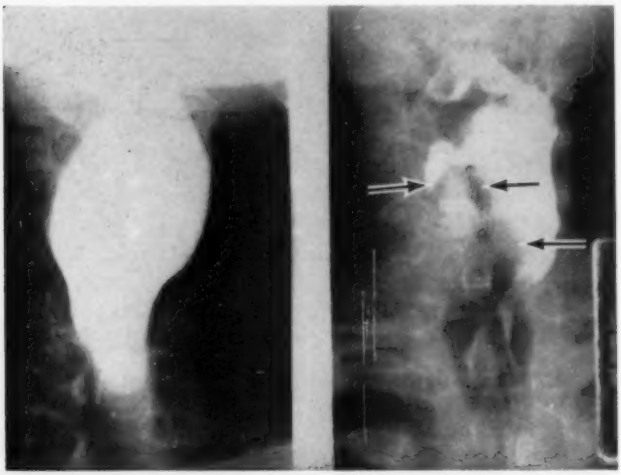

Fig. 7. (H.R.) Barium-filled hypopharynx caught during fluoroscopy in supine position by spot-film (left) rules out a space-occupying mass in right hypopharynx as suggested by Fig. 6, and by the right half of this plate. The latter shows the normally emptied right side of the hypopharynx immediately after completion of swallow, and further supports diagnosis of left-sided pharyngoplegia.

puddled in both pyriform sinuses and no evidence of neoplasm. Patient was discharged without further work-up.

Six weeks later he was re-admitted because of occipital headache of two weeks' duration. Four weeks prior to admission the patient had developed weakness of the left eyelid and left arm. On admission there was ptosis of the left lid, and a dilated, reactionless left pupil; spinal fluid was negative, sputum negative for acid-fast bacilli, WBC 12,000.

Neurological—Patient has a shuffling gait. There is evidence of involvement of Nerves III, IX, and X. Vibratory sensation and heat sensation are increased on the right.

Impression—Tumor of brain stem or metastases to brain stem from a primary tumor in GI tract or lung.

Mirror Examination—Complete paralysis of left vocal cord; puddling of saliva in left pyriform sinus.

X-ray—Ballooning of entire pharynx. Left vocal cord immobile. Ballooning of pharynx probably due to involuntary Valsalva maneuver each time the patient attempts to swallow.

Impression—Left sided bulbar paralysis with inability of the left pyriform sinus to contract, and immobility of the left vocal cord (see Figs. 6 and 7).

During the next six weeks the patient became rapidly worse, with progressive atrophy of the muscles of the neck, shoulder girdle, mandible and both upper and lower extremities. A diagnosis of amyotrophic lateral sclerosis was made before he died, although involvement of Nerves II and III was felt to be against it; autopsy was not obtained.

Discussion—Certain degenerative conditions involving the brain stem may be ushered in by dysphagia and hoarseness as a result of involvement of the nucleus ambiguus with resultant paresis of the constrictor muscles and vocal cords. Very little else may be found initially, even though weakness and asymmetry of the soft palate and posterior pharyngeal wall are carefully looked for.

CONCLUSION.

Isolated pharyngeal paresis may simulate carcinoma of the hypopharynx (pyriform sinus and postcricoid area) because of similar findings on history, physical examination, mirror examination and routine roentgenograms with contrast media. Endoscopic examination having ruled out an intrinsic neoplasm, pharyngeal paresis can best be demonstrated by fluoroscopic comparison of the emptying-time of the two sides of the hypopharynx during barium swallow, preferably in the supine position, and by the demonstration of ballooning and asymmetry of the muscular walls of the hypopharynx during the Valsalva maneuver.

BIBLIOGRAPHY.

1. Stopford, J. S. B.: The Arteries of the Pons and Medulla Oblongata. *Jour. Anat.*, 51:250, 1917.

2. Critchley, M., and Kubik, C.: The Mechanisms of Speech and Deglutition in Progressive Bulbar Palsy. *Brain*, 48:492-534, 1925.

3. Mollison, W.: Dysphagia Due to Pharyngeal Paralysis. *Jour. Laryngol., Otol.*, 43:769, 1928.

4. Furstenberg, A.: An Anatomical and Clinical Study of Central Lesions Producing Paralysis of the Larynx. *Ann. Otol., Rhinol., Laryngol.*, 46, March, 1937.

5. SJOBERG, A.: Experimental and Clinical Studies of Paresis of the Pharynx, Larynx, and Esophagus. *Acta Otolaryngol.*, Suppl. 38, 1939.

6. TILNEY and RILEY: "The Form and Functions of the Central Nervous System," 3rd pd., Paul B. Hoeber, Inc.

7. ELLIOTT, F. A.: Paralysis of the Pharynx. *Jour. Laryngol., Otol.*, 70:331-338, June, 1956.

8. KEOGH, C.: Paralysis of the Pharynx. *Jour. Laryngol., Otol.*, 70:344-351, June, 1956.

9. KRAMER, P.; ATKINSON, M.; WYMAN, S. M., and INGLEFINGER, F. J.: The Dynamics of Swallowing II Neuromuscular Dysphagia of Pharynx. *Jour. Clin. Invest.*, 36:589, 1957.

10. KIRCHNER, J.: "The Motor Activity of the Cricopharyngeus Muscle;" THE LARYNGOSCOPE, 68:1119-1159, July, 1958.

11. COLLEDGE, N.: Discussion of von Eicken: Malignant Disease of the Hypopharynx. *Jour. Laryngol., Otol.*, 47:252, 1932.

12. NEGUS, V.: "The Mechanism of the Larynx." C. V. Mosby Co., 1929.

13. TSCHIASSNY, K.: Personal communication.

14. JACKSON and JACKSON: "Diseases of the Nose, Throat and Ear." W. B. Saunders Co., 1945.

15. MOSHER, H. P.: X-ray Study of Movements of the Tongue, Epiglottis and Hyoid Bone in Swallowing. THE LARYNGOSCOPE, 37:235, April, 1927.

16. TEMPLETON, F. E., and KREDEL, R. A.: The Cricopharyngeal Sphincter: A Roentgenologic Study. THE LARYNGOSCOPE, 53:1, 1943.

17. TEMPLETON, F. E.: "X-ray Examination of the Stomach." University of Chicago Press, 1947.

18. SAUNDERS, J. B. M.; DAVIS, C., and MILLER, E. R.: The Mechanism of Deglutition (Second Stage) as Revealed by Cine-Radiography. *Ann. Otol., Rhinol. and Laryngol.*, 60:897, 1951.

19. BATSON, O. V.: The Cricopharyngeus Muscle. *Ann. Otol., Rhinol. and Laryngol.*, 64:47, March, 1955.

20. BRUNNER, H.: Hypopharyngeal Bar. *Arch. Otolaryngol.*, 61:542, May, 1955.

21. RAMSEY, G. H.; WATSON, J. S.; GRAMIAK, R., and WEINBERG, S. A.: Cine-fluorographic Analysis of the Mechanism of Swallowing. *Radiol.*, 64:498, 1955.

MEDICAL AND SURGICAL TREATMENT OF BELL'S PALSY.*†‡

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The best symptomatic descriptions of paralysis of the motor nerve of the face were those made by Sir Charles Bell before 1841,[1] yet the true causes and the best treatments for non-traumatic facial paralysis still remain controversial, and few patients seek early treatment from the few specialists in this field. In order to understand the rationale for the treatments described in this paper, it is necessary to study some of the probable causes of the sudden, usually transient, paralysis of the VIIth cranial nerve, which has come to be known as "Bell's Palsy."

In the common idiopathic form it is generally believed that there occurs an edema which squeezes the nerve trunk so as to block the passage of neural impulses. Cawthorne[2] has described an "hour-glassing" by either a fibrous band or a congenitally small bony ring at the stylomastoid foramen. He, as well as Sullivan,[3] Collier,[4] Kettel[5] and others, have confirmed the observation of Arthur Duel[6] that there is often a marked bulging of the nerve when its fibrous sheath is slit, in cases of classical "Bell's Palsy."

In my small series of seven operated cases, this was most clear-cut in recent facial paralysis, particularly in recurrent facial paralysis, which tends to show a remarkable improvement within a few hours, or a few days, after surgical decompression. I emphasize patients of recurrent facial paralysis, because whether their paralysis has been on the same side or the opposite side, we have, in these patients, a type

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of control, because all of these patients were decompressed within a few days of the onset of their paralysis. In no case of recurrent "Bell's Palsy" have I seen as slow a recovery as occurred for the bout, or bouts, before decompression, whether the palsy was on the same side or the opposite side. In none of my patients with decompressed "Bell's Palsy" has there yet been a recurrence of facial paralysis after decompression. All of them have been done over one year and the oldest one is of 15 years' duration. Similar statements have been made by Sullivan[3] concerning a much larger series of operated "Bell's Palsy." Apparently, in acute cases, removal of bony and fibrous sheaths of the perpendicular part of the nerve within the mastoid relieves pressures, and thus often leads to recovery from the paralysis; but is pressure on the nerve, *per se*, the primary and only cause of the paralysis?

In 1936, Grunfest[7] reported an experiment with frog nerves and showed that it took 8,000 to 15,000 pounds per square inch to stop nerve impulses when the entire nerve was squeezed in a compression bomb filled with oxygenated mineral oil; however, if there was any bending of axones, neural impulses were reduced or blocked at that point. Perhaps, more important, Lewis and his co-workers,[8] Denny-Brown,[9] and more lately Moldaver,[10] working with clips or tourniquet techniques, have shown that any pressure which interferes with the circulation of the nerve itself, will produce suppression of distal neural impulses; and if the ischemia produced by the pressure is kept up for a sufficient length of time, the palsy will be either long lasting or permanent.

What could instigate a primary ischemia in the facial nerve trunk, leaving aside for the moment, secondary edema and swelling? The story that "Bell's Palsy" often follows a cold draft to the side of the face has been generally accepted by most, since the time of Bell.[1] Although this is vigorously denied by a few, the weight of evidence seems to indicate that a cold draft may precipitate the palsy, in some patients. Not so long ago, Sullivan and Smith incontrovertibly showed that cold can produce facial paralysis in rats,[11] but there are undoubtedly other factors and other precipitating agents, as will be mentioned later.

Let us examine the mechanism of how cold could produce facial paralysis. We have a theory that this might lead, quite logically, to a common denominator, for all "Bell's Palsy" whether associated with virus infections, allergy, arteriosclerosis or what not.

In the mesentery, the conjunctiva of the eye, the chorioallantoic membrane of a chick, the edge of the lung, the spleen and other areas where small blood circulation has been carefully studied, there is no question that chilling will produce local erythrostasis. With slight changes of temperature there are produced contracted arterioles and venules also precapillary closures and, in addition, slow moving stasis, or so-called intravascular agglutinations—the "sludge" of Knisely.[12] With more chilling, platelets and white cells collect in and along the vascular walls, and eventually the circulation in the region is affected by these intravascular elements, as well. This seems to start in the collecting venules, spreads to the capillaries and eventually involves arterioles and arteriovenous shunts, just as does any other local trauma. Clinically, this has often been observed in frostbite of the fingers, toes and ears. Such erythrostasis in the small blood vessels not only results in anoxia of the part with edema and ground substance changes, but also subsequent susceptibility to milder episodes with slight changes in temperature. It is known that similar hypersensitization[13] of nerve tissue can occur, and this may account for recurrent "Bell's Palsy."

Blockage of the small blood vessels with consequent tissue ischemia and secondary edema seems to occur in the VIIth nerve, with many factors other than refrigeration or cooling. There may be an association with allergy or infection, thrombi from other parts of the body and with trauma. I have seen two cases of peripheral facial paralysis which were otherwise identical with so-called idiopathic "Bell's Palsy," associated with perichondritis of the auricle and swollen lymph glands in the upper cervical chain; another case with granuloma of the external canal, which improved after partial removal of the low-grade infectious process near the exit of the nerve. In one case with rubella, the glands were quite large in the region of the stylomastoid foramen, and I have seen another

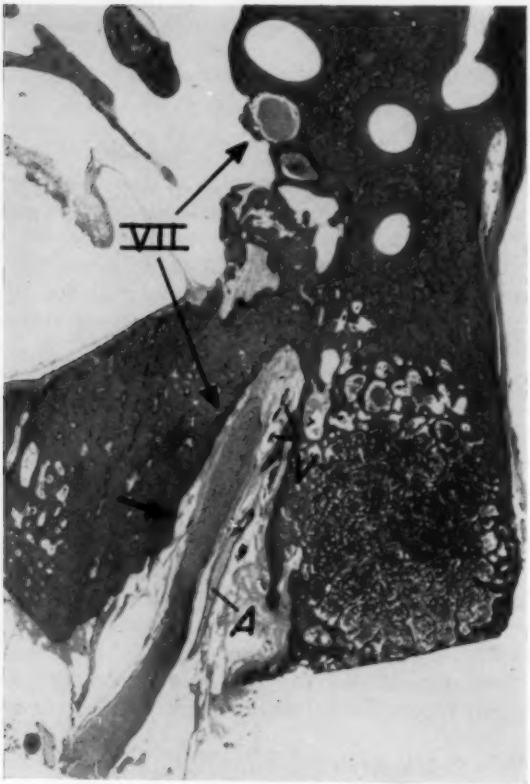

Fig. 1. Vertical section through the temporal bone showing the descending part of the facial nerve (VII) as it goes through a normal stylomastoid foramen; also a cross section of the nerve near the pyramid. Arrow indicates where bony or fibrous narrowing has been observed: A. indicates artery; V. indicates veins.

case with infectious mononucleosis, with glands in the same region.

Sir Charles Bell himself described several cases in which there were abscesses, or large glands in the neighborhood of the stylomastoid foramen.[1] Many times the paralysis occurs in patients with diabetes, or arteriosclerosis. There are, of

course, cases due to injuries from too tight packing in a mastoid wound, or to direct bruising of the nerve.

Most otologists have seen facial paralysis with low-grade and seemingly insignificant concomitant serous otitis, or transudative otitis. It is possible that these symptoms were due to blockage of the stylomastoid artery, which supplies not only the facial nerve but also the mucous membrane of the middle ear (see Fig. 1). I have seen two cases where the bone and the canal were unusually soft but not infected, as described by Kettel.[5]

As mentioned above, apparently anything which could lead to erythrostasis in the vessels in the bony facial canal can produce the palsy. Even Herpes Zoster of the geniculate ganglion must produce a vascular lesion which produces swelling, ischemia and finally blockage of the nerve impulses; however, all these causes of facial paralysis seem to have a common denominator; namely, erythrostasis and ischemia from spasm, intravascular blockage or external pressure involving the small vessels of the nerve trunk. The ischemia causes axone anoxia and secondary tissue edema, which aggravates the situation especially when it can kink the nerve, because anatomical factors such as a fascial collar or a small stylomastoid foramen are present.

With this hypothesis as a background, and considerable experience with Ménière's disease and sudden deafness, which undoubtedly are precipitated by similar vascular phenomena, it was decided to try vasodilation vigorously, and increased circulation treatment techniques for "Bell's Palsy." To date, the same regimen has been used which had proved successful with Ménière's disease; namely, one-tenth of 1 per cent procaine hydrochloride infusions, twice a day, using 250 cc. of a glucose solution, to increase the flow of blood in the small blood vessels, after the patient had been well medicated with nicotinic acid, 50-100 mg. 4 i.d., p.o.,[14] to dilate the small blood vessels.

We have, as yet, treated an insufficient number of patients to prove, definitely, that this or that regimen helps in a disease like "Bell's Palsy." Our results with a small series are

given for what they are worth. It has been variously stated that 55 to 80 per cent of "Bell's Palsy" cases recover without treatment. Thomas[15] in 1955, published a paper in which he gave the comparison of length of time that "Bell's Palsy" took to recover against a regimen of Cortisone, the average was 24 days. From this, and other[16] reviews of the literature, it is difficult to ascertain how the various authors classified their patients with partial, or no improvement. I, therefore, indicated in each group how many had no improvement and how many had partial improvement; how many had full improvement, and the average time in which full improvement seemed to occur. In all, we studied 100 consecutive hospitalized cases, in which there were sufficient notations in the chart for a report to be made. Fourteen were treated with procaine and 86 treated with other methods. In the 14 patients treated with procaine, all had considerable improvement; 12, or 86 per cent, had full improvement, two had partial improvement; the improvement occurred within four to 30 days, making an average of 12.3 days. Of the 86 controls 14, or 14 per cent, had no improvement, another 12, or 14 per cent, had partial improvement, and the remaining 72 per cent had full improvement, ranging from five days to one year, making an average of 76.1 days. Using nicotinic acid alone, there were six patients; two had partial improvement and four had full improvement, in an average of 24.2 days. Seven patients had Cortisone treatment; one remained completely paralyzed; two had partial improvement; four had full improvement, in an average of 24.5 days; thus, the nicotinic acid and Cortisone treated cases seem to show better results than the physiotherapy treated cases; however, the nicotinic acid with intravenous procaine seemed to show the best results in this small series.

Recently we have been treating our cases of sudden deafness with heparin and cumadin, in addition to procaine and nicotinic acid. We have not yet tried this with "Bell's Palsy" but the dramatic improvement in circualtion with heparin, in experimental animals, in addition to that seen with procaine, suggest we should add this medication to our treatment regimen, if we see the patient early enough. For the edema, we

should, perhaps, use Cortisone as well, at least for a few days. In any event, the patient must be hospitalized and the blood chemistry, venous clotting and prothrombin time, carefully controlled. In our hospital this has been supervised by Dr. Stuart W. Cosgriff of the Department of Medicine, and carried out in the same manner as they treat venous thromboses, and as they control hormone therapy afflictions in other parts of the body.

If medical treatment does not produce marked improvement in a "Bell's Palsy" in two months' time it has become our practice to decompress the facial nerve, according to the method of Duel,[6] except that a dental drill, under magnification, is used instead of a hammer and chisel. More and more, electrical studies suggest that two months is too long to wait.

Adequate criteria from electromyograms, chronaxies, and the like, for discovery and evaluation of those patients who will not recover without surgery has been fairly satisfactory in our hands, better prognostic tests are, we believe, urgently needed.

Since the patients selected were almost all hospitalized, and, therefore, likely to be severe cases, perhaps our "Bell's Palsy" sample is skew. This series, however, shows that watchful hoping, heat, massage and electrotherapy do not provide quick and full relief in a sufficient number of cases. Our series compares favorably with the series of Thomas[15] for Cortisone. Nicotinic acid is much cheaper than Cortisone, and much safer. Perhaps one should use Cortisone in large doses for a few days, and then continue with nicotinic acid and procaine. Although the use of i.v. procaine, plus anticoagulants and steroids, twice daily is difficult and somewhat dangerous, I suggest that further trial of such vigorous early treatment is indicated. This paper should stimulate some interest and lead to comparative studies of the relative value of procaine, histamine, steroids, anticoagulants and the like; however, for success, we must persuade all physicians to send in their cases early. We believe, "Bell's Palsy" is a medical emergency like sudden deafness, or any other vascular catastrophe. If we do not start early we can not expect to eliminate the small

blood vessel ischemia and secondary edema. Axone degeneration will begin and theoretically we should resort, within a few days to surgical decompression.

BIBLIOGRAPHY.

1. Bell, Sir Charles: "The Nervous System of the Human Body." Library of Medical and Surgical Science, 1833.

Ibid.: "Medical Classics," Vol. 1. Williams & Wilkens, Baltimore, Sept., 1936.

2. Cawthorne, T.: Peripheral Facial Paralysis; Some Aspects of Its Pathology. The Laryngoscope, 56:653, 1946.

3. Sullivan, J. A.: Recent Advances in the Surgical Treatment of Facial Paralysis and Bell's Palsy. The Laryngoscope, 62:449, 1952.

4. Collier, J.: Facial Paralysis and Its Operative Treatment. *Lancet*, p. 91, July, 1940.

5. Kettel, K.: Bell's Palsy: Pathology and Surgery. *Arch. Otolaryngol.*, 46:427, 1947.

6. Duel, A. B.: Surgical Treatment of Facial Nerve Paralysis. *Acta. Otol.*, 22:373, 1935.

7. Grunfest, H.: "Effects of Hydrostatic Pressures upon the Excitability, the Recovery and the Potential Sequence of Frog Nerve." Cold Spring Harbor Symposia on Quantitative Biol., Vol. V, 1936.

8. Lewis, T.; Pickering, G. W., and Rothschild, P.: Ischemic Paralysis. *Heart*, 16:1, 1931.

9. Denny-Brown, D., and Brenner, C.: Lesion in Peripheral Nerve Resulting from Compression by Spring Clip. *Arch. Neurol. and Psychol.*, 52:1, 1931

10. Moldaver, J.: Tourniquet Paralysis Syndrome. *Arch. Surg.*, 68:136, 1954.

11. Sullivan, J. A., and Smith, J. B.: Otological Concept of Bell's Palsy and Its Treatment. *Ann. Otol., Rhinol. and Laryngol.*, 59:1148, 1950.

12. Knisely, M. H.; Bloch, E. H.; Eliot, T. S., and Warner, L.: Sludged Blood. *Science*, 106:431, 1947.

13. Fowler, E. P., Jr.: Hypersensitization of Tissue to Infections and Allergic Reactions. *Trans. Amer. Acad. Ophthal. and Otolaryngol.*, 59:699, 1955.

14. Fowler, E. P., Jr.: Intravenous Procaine in the Treatment of Mèniére's Disease. *Ann. Otol., Rhinol. Laryngol.*, 62:1186, 1953.

15. Thomas, M. H.: Treatment of Bell's Palsy with Cortisone and Other Measures. *Neurology*, 5:882, 1955.

16. Park, H. W., and Watkins, A. L.: Facial Paralysis: Analysis of 500 Cases. *Arch. Phys. Med.*, 30:749, 1949.

RESULTS OF FENESTRATION AFTER STAPES MOBILIZATION.**

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Otologists who write on the subject of the stapes mobilization operation are unanimous in stating that this procedure does not adversely affect a subsequent fenestration operation.[1-9] This opinion is based upon surgical and acoustical considerations, as well as upon clinical observations.

The clinical evidence reported as empirical validation of the position that the stapes mobilization procedure does not affect the results of a subsequent fenestration involves from one to 45 cases.[1-9] Meurman,[5] who reports on the 45 cases, states that his group had not been subjected to close study. Werth[9] has analyzed 16 cases in terms of average gain as predicted by the Shambaugh formula, but he did not compare these 16 cases to a group on whom the fenestration operation was not complicated by a prior stapes mobilization.

Because considerable clinical importance is attached to the claimed advantage for the stapes mobilization operation, *viz.*, that a subsequent fenestration operation may be performed with no disadvantage in the final result, we felt that a statistical analysis designed to test this hypothesis would be useful.

There are 38 cases in the series of stapes mobilization

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operations performed by one of us (T.E.W.), in which a subsequent fenestration operation was performed. This group of 38 cases was compared with a recent group of 227 fenestration operations[10] that were not complicated by prior stapes mobilization. Hearing level* for speech (spondaic words) is the datum that was used to compare the two groups. Residual hearing was used instead of gain, because residual hearing is the most logical measure of success in this context.[12]

TABLE I.

Mean Hearing Level for Speech Three Months After Fenestration.

	Fenestration after Stapes Mobilization	Fenestration Only
Number of cases	37	227
Mean	26.8	25.7
Standard Deviation	7.97	8.19

TABLE II.

Distribution of Cases as a Function of Hearing Level for Speech Three Months After Fenestration.

	Fenestration after Stapes Mobilization		Fenestration Only	
Hearing Level	%	No.	%	No.
20 db. or better	23.7	(9)	28.2	(64)
21 - 25	29.0	(11)	32.6	(74)
26 - 30	15.8	(6)	18.1	(41)
31 - 35	18.4	(7)	11.9	(27)
36 - 40	2.6	(1)	3.9	(9)
41 db. or worse	10.5	(4)	5.3	(12)
		38		227

First, the two groups were compared with respect to mean hearing level as measured three months after fenestration. The mean hearing level and standard deviation for each group of cases are presented in Table I. The difference between the two groups of 1.1 db. in favor of the uncomplicated fenestration group is not statistically significant ($t = .72$; $p = .47$). One case of stapes mobilization was excluded from this comparison because all hearing was lost after fenestration, and no value could be assigned to hearing level.

*The term "hearing level" is used here instead of the term "hearing loss" as recommended by Davis, Hoople and Parrack.[11]

In addition to the comparison between mean hearing levels for speech for the two groups, we have tabulated the percentage of cases in each group with residual hearing levels for speech of 20 db. or better, 21-25, 26-30, 31-35, 36-40, and 41 db. or worse. These distributions are presented in Table II with the number of cases involved in each percentage enclosed in parentheses. The case of prior stapes mobilization excluded from the comparison between group means, because all hearing was lost after fenestration is included in Table II in the "41 db. or worse" interval. The two groups were compared statistically with respect to the distribution of cases as a function of residual hearing level, and were found to be not different (Chi-Square = 3.17; p = .53).

The results then, of the statistical comparisons between the group of 38 cases in which a stapes mobilization preceded the fenestration operation, and the group of 227 cases for which the fenestration was not complicated by prior stapes mobilization, indicate that the two groups are not different with respect to mean residual hearing level for speech, or with respect to the distribution of cases as a function of hearing level. These results confirm the opinions based upon surgical and acoustical considerations, as well as those based upon clinical experience, that the stapes mobilization procedure does not affect the results of subsequent fenestration.

REFERENCES.

1. Alexander, L. W.: Mobilization of the Stapes in Otosclerosis by a Transtympanic Technique. *Arch. Otolaryngol.*, 66:333-390, 1957.

2. Bellucci, R. J.: Present Status of the Operation for Mobilization of the Stapes. The Laryngoscope, 66:269-292, 1956.

3. Goodhill, V.: Present Status of Stapedolysis (Stapes Mobilization). The Laryngoscope, 66:333-381, 1956.

4. House, H. P.: Personal Experiences with Stapes Mobilization. *Arch. Otolaryngol.*, 65:235-244, 1957.

5. Meurman, Y.: Stapes Mobilization in Otosclerosis. *Arch. Otolaryngol.*, 66:464-479, 1957.

6. Rosen, S., and Bergman, M.: Restoration of Hearing in Otosclerosis by Mobilization of the Fixed Stapedial Footplate. An Analysis of Results. The Laryngoscope, 65:224-258, 1955.

7. Scheer, A. A.: Restoration of Hearing in Otosclerosis by Transtympanic Mobilization of the Stapes. *Arch. Otolaryngol.*, 61:513-534, 1955.

8. Scheer, A. A.: Observation of 500 Cases of Transtympanic Mobilization of the Stapes. *Arch. Otolaryngol.*, 65:245-253, 1957.

9. WERTH, R.: Fenestration After Attempted Mobilization of Stapes (Rosen's Operation) for Otosclerosis. *Acta Oto-laryngol., Stockh.*, 46:70-73, 1956.

10. BILGER, R. C.; GOODMAN, A. C., and WALSH, T. E.: The Fenestration Operation: A Perspective in Time. In preparation.

11. DAVIS, H.; HOOPLE, G., and PARRACK, H. O.: The Medical Principles of Monitoring Audiometry. *A.M.A. Arch. Indust. Health*, 17:1-20, 1958.

12. DAVIS, H., and WALSH, T. E.: Limits of Improvement of Hearing Following the Fenestration Operation. THE LARYNGOSCOPE, 60:273-295, 1950.

OSTEOMA OF THE EXTERNAL AUDITORY CANAL.

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"The large solitary osteoma attached by a pedicle to the bony canal wall is an extremely rare condition."[1] The author was somewhat surprised to find this statement during the course of a literature review in connection with another subject.[2] Four cases observed during the last two years were called to mind. It is the purpose of this paper to define the subject, discuss the treatment, and report these four cases.

DEFINITION.

Dorland's medical dictionary defines an osteoma as any tumor composed of bony tissue. Confusion has arisen clinically[1] between an osteoma of the external auditory canal and the exostosis, which is defined as any bony growth projecting outward from the surface of a bone. By definition, any osteoma projecting outward is also an exostosis. The confusion is increased by the fact that histologically there is no sharp line of distinction (if any) between a true osteoma and many closely related non-neoplastic hyperplasias of bone.[3,4]

From the clinical standpoint it is important that a definition be agreed upon; that the two conditions be considered separately. Osteoma of the external canal refers to the solitary pedunculated bony growth, attached to the tympanosquamous suture superficial to the isthmus (see Figs. 1, 2). It may be attached to the tympanomastoid suture. By contrast exostoses of the external canal are broad-based elevations of bone, usually multiple and bilaterally symmetrical, located deep to the isthmus, at the upper edges of the tympanic bone (see Fig. 3).

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INCIDENCE AND SYMPTOMS.

A number of the current texts[5,6,7,8] do not mention osteoma of the external auditory canal; exostoses are mentioned briefly. Morrison,[9] however, makes a clear-cut distinction between the exostosis and the osteoma, both from the standpoint of location and treatment.

Exostoses of the external canal are common lesions, are

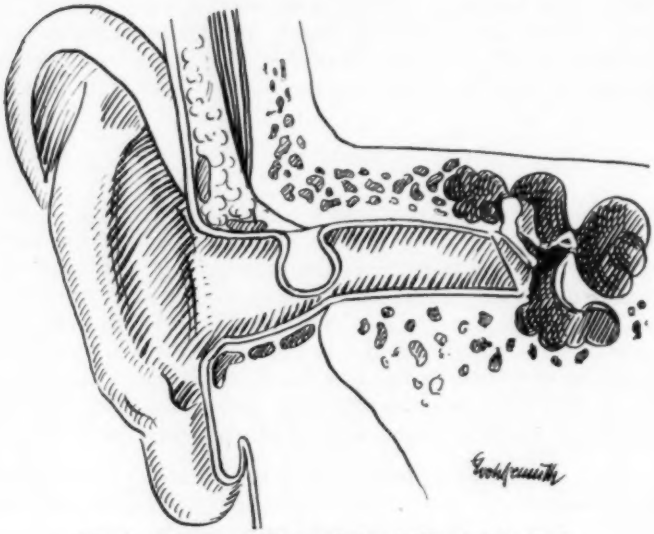

Fig. 1. Osteoma of External Canal (Frontal Section).

usually bilateral and multiple, and rarely demand treatment.[1,5,6,7,8,9] They are seen frequently in the course of examinations for other illnesses, and rarely, if ever, produce symptoms.

The external auditory canal osteoma is always a single unilateral lesion and is considered to be rare. Kline and Pearce[1] reviewed the literature up to 1954, and stated that it was difficult to say how many of the cases reported (over 37) were actually osteomata, and how many were exostoses.

They reviewed in detail the four cases reported during the preceding 15 years, and added a case of their own. They concluded that the condition must be extremely rare judging by the paucity of the reports. Others agree with this conclusion.[10]

Symptoms produced by an osteoma are the result of block-

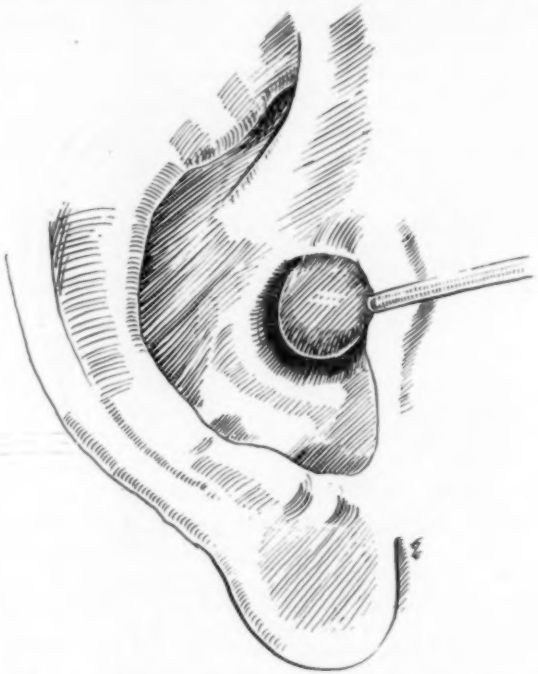

Fig. 2. Osteoma of External Canal (Tragus retracted).

age of the canal. Early there may be excessive wax accumulation deep to the tumor. A complete block may occur after the introduction of water, and eventually hearing loss will develop, due to blockage by collected debris. This often develops before complete occlusion by the tumor. Eventually infection supervenes, accompanied by pain. Initially the in-

fection is limited to the external canal, but it may spread to the middle ear and mastoid. A long-standing occlusion and infection will result in finding a situation not unlike a cholesteatoma, with extensive destruction of the mastoid and middle ear, necessitating radical mastoidectomy.[1]

TREATMENT.

A radical or modified radical mastoidectomy is necessary in an occasional case, to treat adequately the temporal bone

Fig. 3. Exostoses of External Canal.

suppuration secondary to the blockage by the tumor. This was true in two of the five cases reviewed by Kline and Pearce.[1] It would seem that modified radical mastoidectomy could have been avoided in two of the cases reviewed by them. A simpler (transmeatal) procedure might have been more satisfactory for the patient.

Treated early, that is before complications develop, and with an understanding of the anatomy involved, the majority of osteomata may be removed transmeatally. An endaural

incision may be necessary to facilitate exposure. The procedure found most satisfactory in the author's experience is as follows:

General anesthesia, with an endotracheal tube, is used. The ear is prepared as for mastoidectomy. If the outer third of the canal is wide, and the tragus is not excessively prominent, no incision will be necessary. Otherwise the upper arm of an endaural incision is made and a retractor inserted. A sturdy attic hook or ear curette is inserted past the edge of, and deep to the osteoma (and is later used to deliver the tumor). This can usually be slipped deep to the tumor, even where no opening is visible. In an occasional case, posterior canal wall bone may advantageously be removed before inserting the hook. An assistant holds this hook. A curved gouge is then directed at the pedicle and tapped lightly, loosening the tumor. It is lifted out with the hook or curette. It may be necessary to curette lightly or burnish the canal wall at the site where the tumor was attached.

A small area of canal wall is left denuded. If this is less than one-third of the circumference, as it usually is, a skin graft will not be necessary. If greater than one-third of the circumference, it is wise to enlarge the canal with the bur, and apply a thin full-thickness skin graft removed from behind the ear. The canal is packed with a finger cot filled with vaseline gauze, and a mastoid dressing is applied. Any endaural incision is, of course, sutured.

Antibiotics are given pre- and post-operatively. The packing is removed on the third day, and a dry (powder) treatment is utilized thereafter until healing takes place.

One last word on treatment: an osteoma of the external canal as herein defined should be removed, even though not causing symptoms. This tumor is in a position where it is continually exposed to trauma. Continued growth of the tumor may result in complications necessitating radical mastoid surgery. Surgery undertaken after the onset of complications is never so satisfactory.

CASE REPORTS.

Case 1. A 25-year-old white male was referred because of a two-year history of a gradually enlarging growth in the right ear canal. Wax and water would collect deep to it. Examination showed a bony tumor attached to the anterior superior bony wall, superficial to the isthmus, filling all but one mm. of the canal diameter. Hearing was normal.

Under endotracheal anesthesia the upper arm of an endaural incision was made to facilitate exposure. A small dull ear curette was passed deep to the lesion, which was seen to be attached by a broad pedicle to the tympanosquamous suture. It was dislodged by a firm blow on a curved gouge, the blade of which had been placed at the pedicle, and was delivered with the curette. The tympanic membrane was normal. The incision was closed and the canal was packed with a finger cot filled with vaseline gauze. A mastoid dressing was applied. The dressings were removed in three days, and the canal was healed in two weeks.

Case 2. A 22-year-old white male was referred for treatment of recurrent otitis externa. Examination revealed a subacute right external otitis, due apparently to vigorous attempts by the patient to remove wax from the ear. There was a small (3 mm.) pedunculated bony tumor attached antero-superiorly at the junction of the bony and cartilaginous canal.

Removal of the tumor was advised after the otitis had subsided. The patient refused treatment.

Case 3. A 27-year-old Negro male was referred because of a one-and-a-half year history of intermittent discomfort in the left ear. Hearing loss had been constant, and amounted to 40 dbs. (conductive). Examination revealed a complete block of the left external canal by a bony growth which was attached by a pedicle to the posterior superior canal wall superficial to the isthmus.

Under endotracheal anesthesia an S-shaped endaural incision was made. The membranous canal was separated for a short distance to the pedicle, and cut to this point. An external cuff of membranous canal was then fashioned, based inferiorly, and turned anteriorly. With a bur some of the posterior canal wall was taken down until the pedicle could be seen clearly. A sturdy attic hook was then passed deep to the osteoma. The tumor was dislodged, using a mallet and curved gouge, and delivered with the hook. There was much epithelial debris impacted beneath this. The tympanic membrane was normal. The upper arm of the endaural incision was closed, the external cuff was thinned and replaced, and the canal was packed with a finger cot filled with vaseline gauze. A mastoid dressing was applied.

The dressing and packing were removed in three days, and the canal was healed in three weeks. Post-operative audiogram revealed normal hearing.

Case 4. A 23-year-old white male was referred because of a four-month history of wax impacted in the right ear, with subsequent infection of the canal. Examination revealed a pedunculated bony growth, attached antero-superiorly, to the bone of the right external canal, superficial to the isthmus, with less than one-third of the normal lumen remaining. The hearing was normal despite a large plug of cerumen.

The osteoma was removed under endotracheal anesthesia.* A small sturdy attic hook was placed deep to the lesion. A tap on a curved gouge, the blade of which had been placed at the pedicle, resulted in separation

*Surgery performed by Colonel Edward J. Whiteley.

of the tumor. It was delivered with the hook. There was no bleeding. The tumor had been attached to the tympano-squamous suture. The cerumen plug was removed, revealing a normal tympanic membrane. Gelfoam was placed in the canal and removed in one day. The canal healed within a week.

SUMMARY.

An osteoma of the external auditory canal, although not common, is not extremely rare. It must be differentiated from multiple exostoses, which are common. These two lesions have been defined and discussed from the standpoint of symptomatology and treatment.

An osteoma of the external auditory canal is always a surgical problem. Four cases are reported.

REFERENCES.

1. KLINE, O. R., and PEARCE, R. C.: Osteoma of the External Auditory Canal. *Arch. Otolaryngol.*, 59:588-593, 1954.

2. SHEEHY, J. L.: "Present Day Temporal Bone Surgery." The Medical Bulletin of the United States Army, Europe, Feb., 1958.

3. MOORE, R. A.: "A Textbook of Pathology." W. B. Saunders Co., Phila., 1944.

4. BOYD, W.: "Surgical Pathology," 6th Ed. W. B. Saunders Co., Phila., 1948.

5. JACKSON, C., and JACKSON, C. L.: "Diseases of the Nose, Throat and Ear." W. B. Saunders Co., Phila., 1945.

6. BOIES, L. R.: "Fundamentals of Otolaryngology," 2nd Ed. W. B. Saunders Co., Phila., 1954.

7. BALLANGER, H. C.: "A Manual of Otology, Rhinology, and Laryngology," 3rd. Ed. Lea & Febiger, Phila., 1947.

8. WARD, G. E., and HENDRICK, J. W.: "Tumors of the Head and Neck." The Williams & Wilkins Co., Balt., 1950.

9. MORRISON, W. W.: "Disease of the Ear, Nose, and Throat." Appleton-Century-Crafts, Inc., New York, 1948.

10. SELIGMAN, E., and SELIGMAN, M.: Osteoma of the External Auditory Canal; Case Report. THE LARYNGOSCOPE, 66:1592-1594, 1956.

AN ELECTROPHYSIOLOGIC PROCEDURE FOR DETERMINATION OF AUDITORY THRESHOLD IN CHILDREN.*

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Several electrophysiologic indices have been used in clinical audiometry in the attempt to determine auditory thresholds in adults and children who cannot or will not respond adequately during conventional audiometric tests which use conditioned behavioral responses. The most commonly used electrophysiologic index is some change in electric characteristics of the skin in response to auditory stimulation (electrodermal response or EDR). This change, presumably mediated primarily by the activity of the sweat glands of the skin, can be demonstrated by alterations in impedance to flow of electric current or by alterations in electric potential between two points on the skin. Another index employed clinically is a change in the pattern of electric activity of the brain in response to auditory stimulation (electroencephalic response, or EER).

The principal value of auditory tests which use EDR and EER in response to sound as indicators of "hearing" is the information which they can contribute beyond that to be derived from observing behavioral responses. Consequently, it is desirable that judgment of a change in an electrodermogram, or an electroencephalogram, as a response to a particular auditory stimulus be independent of the tester's knowledge of the stimulus and of the patient's overt behavior during stimulation. It is also desirable that threshold be estimated

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from the distribution of responses on the basis of predetermined criteria, just as it is in the more conventional audiometric procedures.

The procedure developed by Stewart[13] and elaborated clinically by Doerfler and McClure[5] satisfy the conditions mentioned in the previous paragraph. Their procedure, however, requires some prior knowledge of a patient's auditory sensitivity in order, *1.* to find a level at which a stimulus is definitely audible for a preliminary conditioning period, and *2.* to establish a limited range of intensities within which their auditory stimuli can be randomly ordered. Consequently, Doerfler and McClure did not determine thresholds independent of any knowledge of behavioral responses; they confirmed that threshold lay within a range previously established by behavioral responses.

Preliminary determination of an approximate threshold is usually difficult with the particular adults for whom electrophysiologic tests are most necessary. It is even more difficult with very young children, and with children who are mentally retarded, emotionally disturbed, or aphasic.

The purpose of this paper is to describe another procedure for determination of auditory thresholds using both EDR and EER as indicators of "hearing," and to report results of attempts to use this procedure to assess the hearing of some children with auditory disorders.

SUBJECTS.

The subjects for this study were drawn from the children enrolled in full-time classes at Central Institute for the Deaf (CID). These children represent a calibrated sample, in a sense, because their hearing has been evaluated yearly since their enrollment at CID.

*Apparatus.**

The apparatus used in this study was described by Charan and Goldstein.[2] The stimulating apparatus consisted of an

*The basic apparatus was a gift of the Children's Research Foundation, St. Louis, Mo.

audiometer and a device for applying electric shock. Sound was delivered monaurally either through earphones held in place by a headband, or through hearing-aid type earphones held in place in the ear canal with the children's own earmold. If the child did not have his own earmold, a stock-mold was used, and it was taped in place to minimize acoustic leaks and to prevent its falling out of the canal. It is preferable

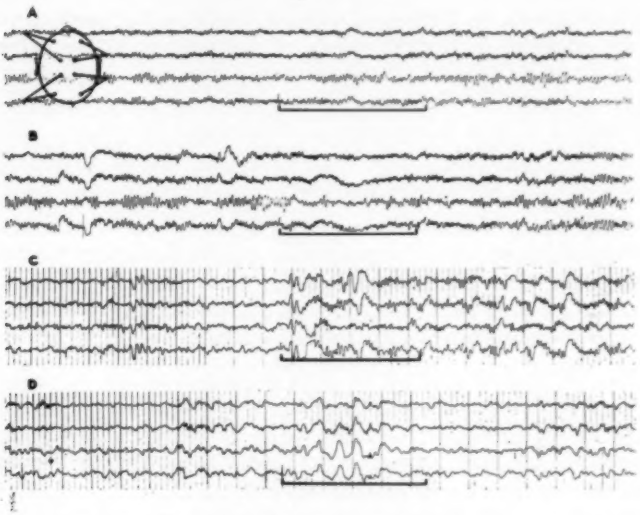

Fig. 1. Sample electroencephalograms from the record of subject D during his EER-Audio. The line under each strip indicates the duration of the auditory stimulus (approx. 5 sec.). A and B: Early stages of test, child awake; C and D: child asleep. Note clear electroencephalic responses in B, C and D.

to use the hearing-aid type earphone in the EER-Audio portion (to be described) during which the child is asleep. These smaller earphones, without a headband, are less likely than the larger earphones, *1.* to interfere with the comfort of the child's head during sleep; *2.* to interfere with the EEG electrodes attached to the child's scalp, and *3.* to loosen and to allow acoustic leaks.

The recording apparatus was a Grass four-channel electro-

encephalograph. The electroencephalogram (EEG) was recorded by conventional procedures.[6] The usual electrode placements were varied, however, according to a suggestion by Derbyshire,[3] so that the parietal lead was placed slightly posteriorly and the temporal lead was placed in a position in front of the usual parietal location. The central leads from both sides were, therefore, bunched closely together near the top of the head. Fig. 1 shows the placement of the electrodes as well as a sample of the kind of recordings obtained from them. The electrodes were attached to the head on portions of the scalp previously cleaned with alcohol. Bentonite paste[6] insured satisfactory contact between the electrode and the scalp. The electrodes were held in place with adhesive tape.

The electrodermogram (EDG) was recorded on one channel of the electroencephalograph. This method of recording has been described by Mays,[10] and by Goldstein, Ludwig and Naunton.[9] In the present study, recording electrodes were taped to the tips of two fingers of the left hand. The shock electrodes were attached to the calf of the left leg.

PROCEDURE.

EER-Audio.[8]

The EER-Audio is performed when a child is asleep. It is desirable that the children be tested in the evening or during regular nap-time, so that advantage can be taken of natural sleep whenever possible. When a child does not go to sleep naturally, he can be lightly sedated. The children in this study were given Seconal or Nembutal orally, under a neurologist's supervision.

The test consists of 96 stimulus-conditions divided into six similar series of 16. A sample series is shown in Fig. 2. Six different series are used to prevent inadvertent memorization of the sequence of stimuli in any series. The order in which the six series are presented to any child is also randomized. The stimuli are tones of 500 c.p.s. or 2000 c.p.s. with a duration of five seconds. Each frequency is presented at three different intensities corresponding to the audiometric levels of 10, 50 or 90 db. hearing-loss. If his general be-

havior indicates that a child has normal or nearly normal auditory sensitivity, the range of intensities can be restricted to 10, 40 and 70 db. hearing-loss. If there is any doubt whatsoever, the broader range of 10, 50 and 90 db. hearing-loss is used.

The combination of two frequencies and three intensities in both ears makes a total of 12 stimuli. Randomly scattered among these 12 stimuli are four control-intervals, *i.e.*, four

No.	Ear	Frequency (in c.p.s.)	Hearing-Loss (db.)
1	R	2000	10
2	L	2000	90
3	R	2000	90
4	L	2000	10
5	L	500	90
6		CONTROL	
7	L	500	10
8	L	500	50
9	L	2000	50
10		CONTROL	
11	R	500	90
12		CONTROL	
13	R	2000	50
14	R	500	10
15		CONTROL	
16	R	500	50

Fig. 2. One of the series of stimuli used in the EER-Audio. The other five series have identical stimuli but in different sequences.

times a stimulus-mark is recorded on the electroencephalogram when no auditory stimulus is presented to the child. A number corresponding to the order of the stimulus or control in the pre-arranged schedule is placed by each stimulus-mark. A minimum of 25 seconds is allowed to elapse between stimuli. It is not always possible to complete all six series. When a child awakes and will not go back to sleep or relax quietly, the test is discontinued.

The records are not analyzed until after the test is completed.

EDR-Audio.[8]

The EDR-Audio is performed when the child is awake, either in the morning while he is alert, or in the afternoon after he has napped.

This test, like the EER-Audio, consists of 96 stimuli, divided into six similar random series of 16 stimuli; and the stimuli are 500 c.p.s. or 2000 c.p.s. tones of five seconds duration (see Fig. 3). Each frequency is presented at four different intensities corresponding to the audiometric levels 20, 40, 60 and 80 db. hearing-loss. Two frequencies and four intensities in both ears make a total of 16 stimuli for each series. It is not necessary to use controls in the EDR-Audio because of

No.	Ear	Frequency (in c.p.s.)	Hearing-Loss (db.)	Shock
1	L	500	20	
2	L	500	40	
3	R	2000	80	+
4	R	500	80	
5	R	2000	60	+
6	R	500	40	+
7	L	2000	20	+
8	R	2000	40	
9	L	2000	40	+
10	L	500	60	
11	R	500	60	+
12	L	2000	80	+
13	R	2000	20	
14	L	2000	60	
15	L	500	80	
16	R	500	20	+

Fig. 2. One of the series of stimuli used in the EDR-Audio. The other five series have identical stimuli but in different sequences.

the infrequency of random changes with the appropriate latency, time-course, and amplitude, which may be mistaken for responses to auditory stimuli.

One-half of the 16 stimuli are followed by annoying electric shocks, regardless of the intensity of the auditory stimulus. Because it is not required that shocks follow tones which are unquestionably audible, there is no need to have prior knowledge of an approximate hearing-loss. We do not have certain knowledge as to what effect the use of shocks with inaudible tones will have on conditioning or responsiveness in general. We have had some experience with adults, however, which indicates that shocks not preceded by audible tones do not depress responsiveness appreciably.

As in the EER-Audio, only a number is placed by the

stimulus-mark corresponding to the order of the stimulus in the pre-arranged schedule. The records are analyzed after the test is finished.

The EER-Audio is usually administered first, because the test involves no discomfort for the child, and he is not reluctant to return for the EDR-Audio. Generally, it is more difficult to secure the cooperation of a child for the EER-Audio after he has been subjected to the shocks in the EDR-Audio.

ANALYSIS OF THE RECORDS.

The records are analyzed after the electrophysiologic tests are completed. The only marks on the electroencephalograms and electrodermograms other than the tracings themselves are; *1.* the various stimulus-marks, and *2.* a number alongside of each mark corresponding to the order of the tone or control in the pre-arranged schedule. The tester can note in a separate protocol the overt behavior of a child in response to a stimulus. Judgments of responses to all 96 stimuli, or controls, are made before referring to the schedule.

EER-Audio.

Judgment of the presence of an electroencephalic response to a tone depends solely on how much the EEG pattern in or following the five seconds during which the tone is presented differs from the preceding pattern. Derbyshire and his coworkers[4] have described the kinds of changes which can occur in response to a tone. Fig. 1 shows samples of electroencephalic responses from one of the children in this study (Subject D).

Although not all responses can be judged with equal certainty, a simple "yes" or "no" decision is made for each stimulus. Responses to the 96 stimuli, or controls, are tabulated according to the scheme shown in Figs. 4-10.

EDR-Audio.

Judgment of the presence of an electrodermal response to a tone depends primarily on the latency of a change in the

impedance or potential of the skin following the onset of the tone. The average latency is very close to 2.1 seconds. Changes with latencies less than 1.5 seconds, or greater than 3.0 seconds, are disregarded unless a child consistently responds outside this range. Changes of impedance or potential within this period usually are larger than the random changes, and usually reach maximum in a shorter time than do the random changes. Responses to the 96 stimuli are tabulated in the same way as described for the EER-Audio.

ESTIMATION OF THRESHOLD OR HEARING-LOSS.

In a plot of behavioral responses as a function of intensity elicited by conventional audiometric procedures, one ordinarily finds a point of zero responses, a point of 100 per cent

TABLE I. CRITERIA FOR ESTIMATION OF THRESHOLD.

Number of false-positives in 24 control-intervals.	Number of responses to six stimuli to be significantly* more than false-positives.
0	2
1	3
2	4
3	5
4	6
5	7
6	8
7	9
8	10

*5 per cent level of a one-tailed distribution.

responses, and a sigmoid curve describing the locus of the intermediate points. The 50 per cent point is usually chosen as threshold.

The curve describing the frequency of electrophysiologic responses as a function of the intensity of the auditory stimulus usually does not have a zero point or a 100 per cent point, and the locus of the intermediate points is not certain. A practical zero can be established for each individual; however, from the relative number of false-positives in the control-intervals. The false-positives may be the result of faulty judgment during analysis of the records, or the result of an actual response to some extraneous stimulus. Experience has shown that the number of false-positives in the EDR-

Audio is small; thus, practical zero is not very different from true zero. In the EER-Audio, the percentage of false-positives is somewhat larger.

Statistical calculations[12] provide a criterion for determining the stimulus-level at which significantly more responses than false-positives occur (see Table I). This criterion is based on the 5 per cent point for a one-tailed test. The weakest stimulus-level at which this criterion is met is regarded as (or above) the subject's threshold.

Because there are no control-intervals in the EDR-Audio, estimates of threshold are made conservatively. One can assume that there would have been a maximum of two or three false-positives if 24 control-intervals had been used. Thus, in the EDR-Audio there must be at least three responses to six stimuli at a given level to indicate "hearing" at that level.

The following section describes the results of attempts to confirm the above criteria for threshold. For this purpose we have chosen some children from the full-time classes at Central Institute for the Deaf, who have been tested repeatedly by conventional audiometry, and have shown the same audiogram over a period of several years. We shall also describe the attempts to apply these criteria to the determination of hearing-losses in some children for whom conventional procedures produced inadequate or conflicting results. Although the audiometric records were on file, we had no knowledge of the audiograms of any of the children before they were tested. The results of the electrophysiologic and the conventional tests were compared only after the records from the electrophysiologic tests had been analyzed.

RESULTS.

The following examples will show how the application of the above criterion served to determine the hearing-loss within ± 10 db. of thresholds previously determined by conventional audiometry.

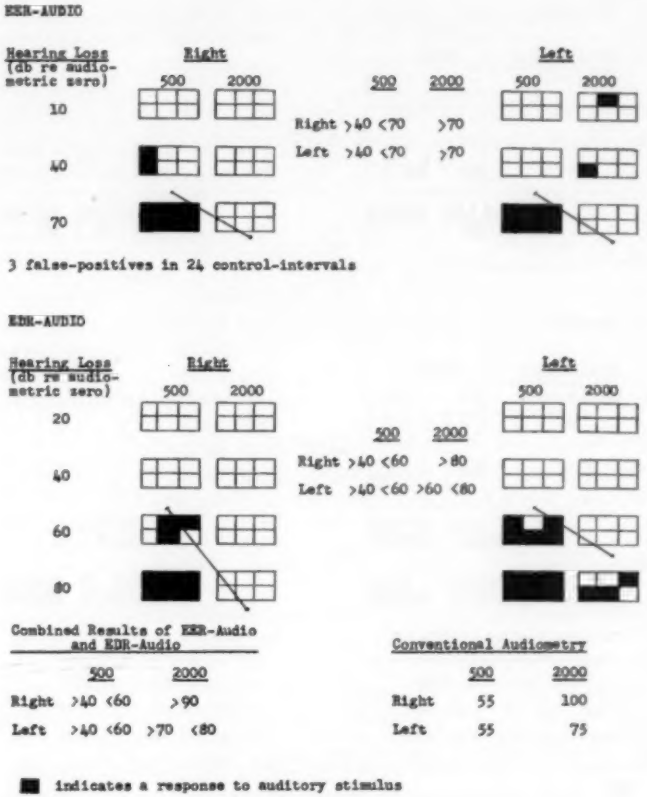

Fig. 4. Plot of responses from the EER-Audio and EDR-Audio for Subject A.

A. 13 years old (see Fig. 4).

Because a relatively small hearing-loss was suspected on the basis of general behavior, the hearing-losses of 10, 40 and 70 db. (re: audiometric zero) were used in the EER-Audio instead of 10, 50 and 90 db. ordinarily recommended.

There was not sufficient intensity at 70 db. hearing-loss

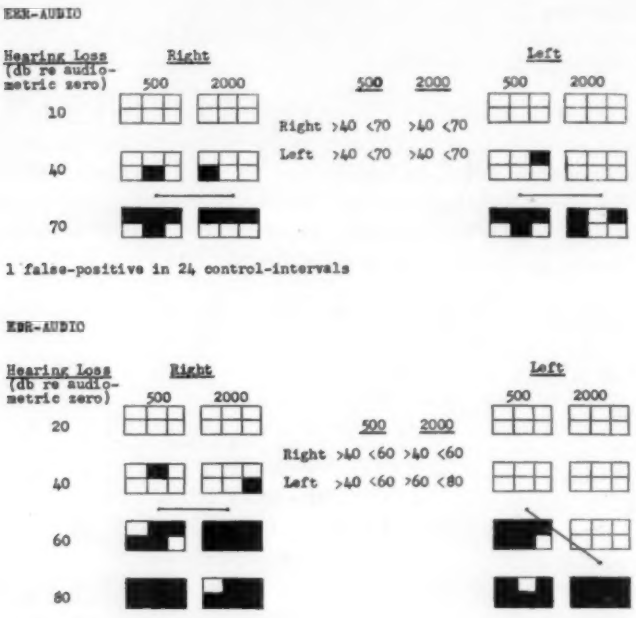

Fig. 5. Plot of responses from the EER-Audio and EDR-Audio for Subject B.

to elicit three responses to 2000 c.p.s. delivered to either ear. The responses to 500 c.p.s. were sufficiently frequent so that on the basis of the EER-Audio alone, this child's hearing-losses were:

	500	2000
Right	>40 <70 db.	>70 db.
Left	>40 <70 db.	>70 db.

According to the EDR-Audio his hearing-losses were:

	500	2000
Right	>40 <60 db.	>80 db.
Left	>40 <60 db.	>60 <80 db.

The combined results of EER-Audio and EDR-Audio indicated the hearing-losses to be:

	500	2000
Right	>40 <60 (55) db.	>80 (>100) db.
Left	>40 <60 (55) db.	>70 <80 (75) db.

The numbers in parentheses are the hearing-losses determined by conventional audiometry by other examiners with other equipment.

B. 11½ years old (see Fig. 5).

Again, because only a moderate hear-loss was suspected, intensities corresponding to 10, 40 and 70 db. hearing-loss were used. On the basis of our criterion, the child's hearing-losses according to the EER-Audio were:

	500	2000
Right	>40 <70 db.	>40 <70 db.
Left	>40 <70 db.	>40 <70 db.

According to the EDR-Audio the hearing-losses were:

	500	2000
Right	>40 <60 db.	>40 <60 db.
Left	>40 <60 db.	>60 <80 db.

The combined results of the EER-Audio and EDR-Audio indicated the hearing-losses to be:

	500	2000
Right	>40 <60 (50) db.	>40 <60 (50) db.
Left	>40 <60 (50) db.	>60 <70 (55) db.

The numbers in parentheses again represent the hearing-losses from conventional audiometry performed by other examiners with other equipment. The discrepancy (approximately 10 db.) between the thresholds determined electrophysiologically and psychophysically for 2000 c.p.s. in the left ear is not appreciably more than can be expected from differences in calibration between the two audiometers or from test-retest reliability.

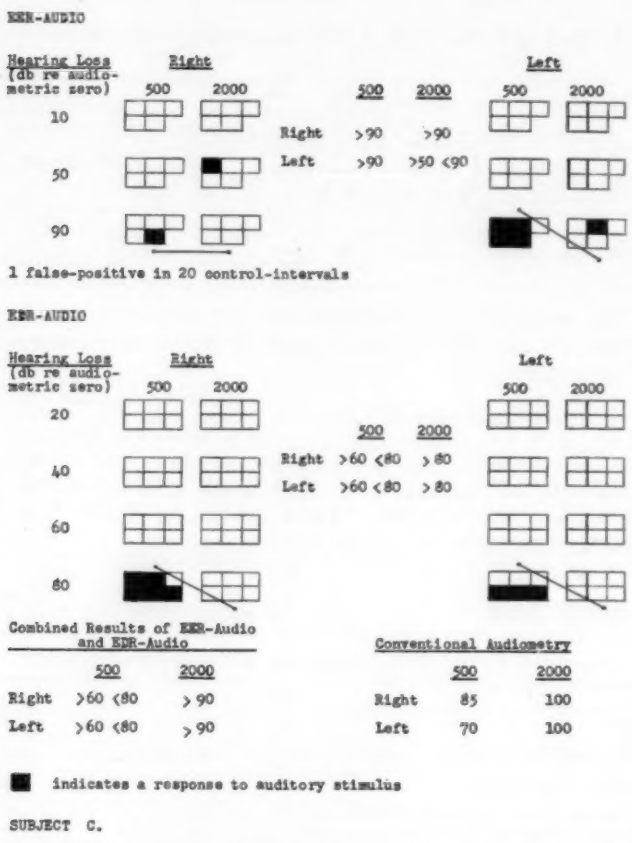

Fig. 6. Plot of responses from the EER-Audio and EDR-Audio for Subject C.

C. 7½ years old (see Fig. 6).

Only five instead of the usual six series were given in the EER-Audio, because this child awoke before the end of her test. On the basis of three responses to five stimuli as the criterion for "hearing," this child's hearing-losses were:

	500	2000
Right	>90 db.	>90 db.
Left	>50 <90 db.	>90 db.

The six series of the EDR-Audio were completed. The hearing-losses according to this test were:

	500	2000
Right	>60 <80 db.	>80 db.
Left	>60 <80 db.	>80 db.

The EDR-Audio showed "hearing" in the right ear for 500 c.p.s. at the 90 db. level; the EER-Audio did not.

The following are the hearing-losses based on both the EER-Audio and EDR-Audio combined, along with the findings from conventional audiometry (in parenthesis):

	500	2000
Right	>60 <80 (85) db.	>90 (>100) db.
Left	>60 <80 (70) db.	>90 (100) db.

The discrepancy (approximately 10 db.) between the findings determined electrophysiologically and psychophysically for 500 c.p.s. in the right ear is not appreciably greater than differences in calibration or test-retest reliability.

D. 10 years old (see Fig. 7).

On the basis of our criteria, this child's hearing-losses according to the EER-Audio were:

	500	2000
Right	>10 <50 db.	>50 <90 db.
Left	>50 <90 db.	>90 db.

According to the EDR-Audio his hearing-losses were:

	500	2000
Right	>40 <60 db.	>80 db.
Left	>60 <80 db.	>80 db.

The hearing-losses based on both tests and the results of conventional audiometry (in parentheses) are as follows:

	500	2000
Right	>40 <50 (35) db.	>80 <90 (95) db.
Left	>60 <80 (75) db.	>90 (>100) db.

There are two minor discrepancies (approximately 10 db.) between electrophysiologically and psychophysically determined thresholds for 500 c.p.s. and 2000 c.p.s. in the right ear. As in the cases previously cited, these discrepancies can

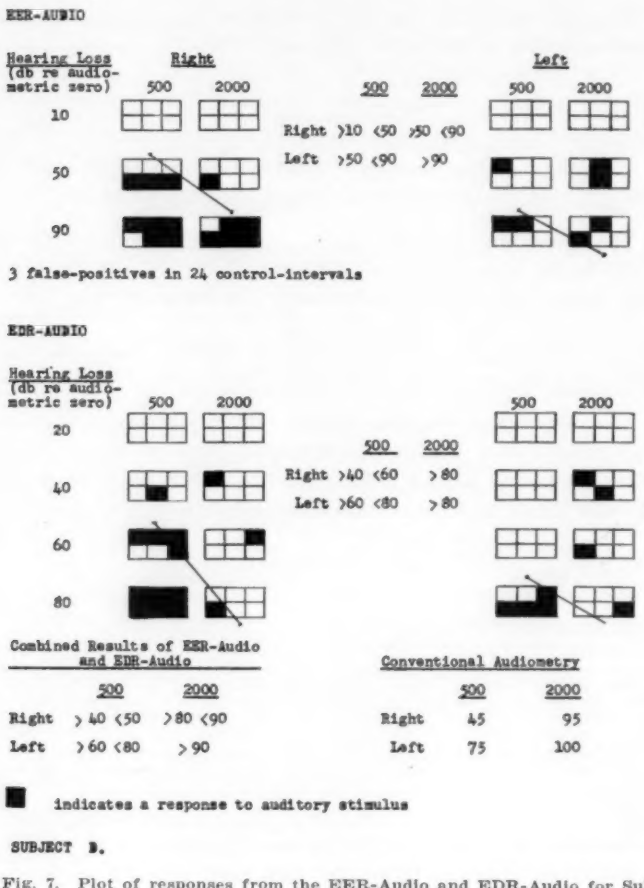

Fig. 7. Plot of responses from the EER-Audio and EDR-Audio for Subject D.

be accounted for by differences in calibration or by test-retest reliability.

The preceding "calibrated" examples not only confirm a criterion for threshold derived from theoretical considerations, but also allow an estimation of the frequency with which judgments of false-positives are made. In the EER-

Audio of each of the preceding instances, there were 3 in 24, 1 in 24, 1 in 20, and 3 in 24, respectively, false-positives in the control-intervals. If stimuli below each child's threshold are considered as controls, a similar frequency of false-positives is noted: 4 in 60, 3 in 48, 2 in 60, and 6 in 48, respectively. On the basis of these four examples, one may expect anywhere from 4 to 13 per cent false-positives with the EER-Audio.

In the EDR-Audio, these same children gave the following number of false-positives to stimuli below their thresholds: 0 in 66, 2 in 48, 2 in 84, and 8 in 78, respectively. On the basis of these four children, an observer should expect to judge from 0 per cent to 10 per cent false-positives in the EDR-Audio. This is comparable to about 2 per cent false-positives found in another study on normal adults.[12]

The data reported here is typical of that found in successful tests with cooperative patients, but it should be emphasized that the EER-Audio and EDR-Audio are not always so successful as in the four cases just described.

In the following examples, varying degrees of success were achieved, and certain modifications in the criterion for threshold were made in order to salvage some usable information from the tests.

E. 8 years old (see Fig. 8).

Because there were no false-positives in the 24 control-intervals of the EER-Audio, only two responses to six stimuli were required to establish threshold. On this basis the hearing-losses for this child were:

	500	2000
Right	>50 <90 db.	>90 db.
Left	>50 <90 db.	>90 db.

In the EDR-Audio there were no definite EDRs to any of the 96 stimuli. What is plotted in the analysis of the EDR-Audio in Fig. 8 were the only definite overt responses observed during the test. On the basis of these overt responses, the hearing-losses were:

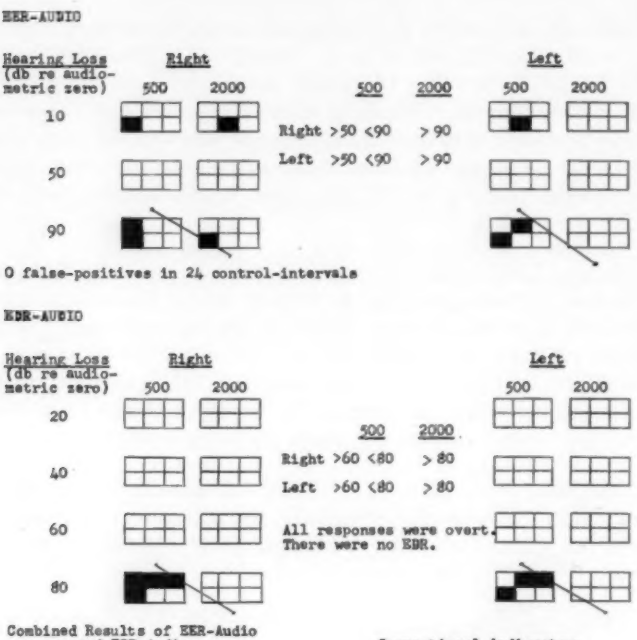

Fig. 8. Plot of responses from the EER-Audio and EDR-Audio for Subject E.

	500	2000
Right	>60 <80 db.	>80 db.
Left	>60 <80 db.	>80 db.

On the basis of the evidence from both tests, the hearing-losses were estimated to be:

	500	2000
Right	>60 <80 db.	>90 db.
Left	>60 <80 db.	>90 db.

Because the range of hearing-losses from the EER-Audio coincided with the range of hearing-losses estimated from overt responses in the EDR-Audio, the hearing-losses could be reported with considerable certainty. The findings allowed us to resolve a difference in educational recommendations based partially on differing pure-tone audiograms. Initially it was believed that this child had only a mild hearing-loss, and it was recommended that he be educated as an aphasic child. His educational development, however, was like that of a deaf child; and later audiograms determined by conventional audiometry, though they had been reported with qualifications, were confirmed by the above estimate of hearing-loss from the electrophysiologic tests.

F. 8 years old (see Fig. 9).

In the EER-Audio there were at least three responses to six stimuli only for 2000 c.p.s. in the right ear, at 50 db. hearing-loss; there was only one response at 90 db. hearing-loss. To the six stimuli of 2000 c.p.s. at 50 db. hearing-loss in the left ear, there were two responses; at all other frequencies and intensities there were one or no responses. One might have assumed from the pattern of responses that the hearing-losses were:

	500	2000
Right	>90 db.	>10 <50 db.
Left	>90 db.	>10 <50 db.

Such a pattern, however, is rarely, if ever, seen clinically and in this instance was not consistent with the child's usual reactions to sound. In addition, the total of 11 responses to all 96 stimuli is not significantly more than the two false-positives in the 24 control-intervals. Our assumption, therefore, was that the child's hearing-loss was greater than 90 db. for 500 c.p.s. and 2000 c.p.s. in both ears.

In the EDR-Audio there were no responses to the 96 stimuli used either because this child was particularly difficult to condition, or because the sound was not sufficiently intense to elicit responses. We tested these possibilities at the end of the regular EDR-Audio by presenting sounds exceeding the

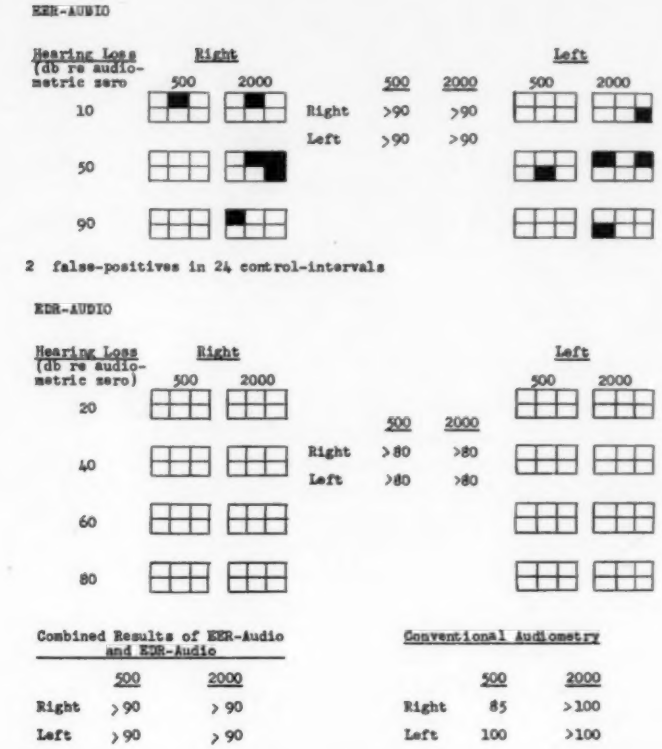

Fig. 9. Plot of responses from the EER-Audio and EDR-Audio for Subject F.

intensity for 90 db. hearing-loss. The child consistently gave EDRs to these latter sounds.

The lack of electrophysiologic responses below the 90 db. hearing-loss was in accord with his limited reaction to sound from the group hearing-aid in his classroom. The findings were also in accord with conventional audiometry (reported with limited certainty), and helped to resolve a difference in

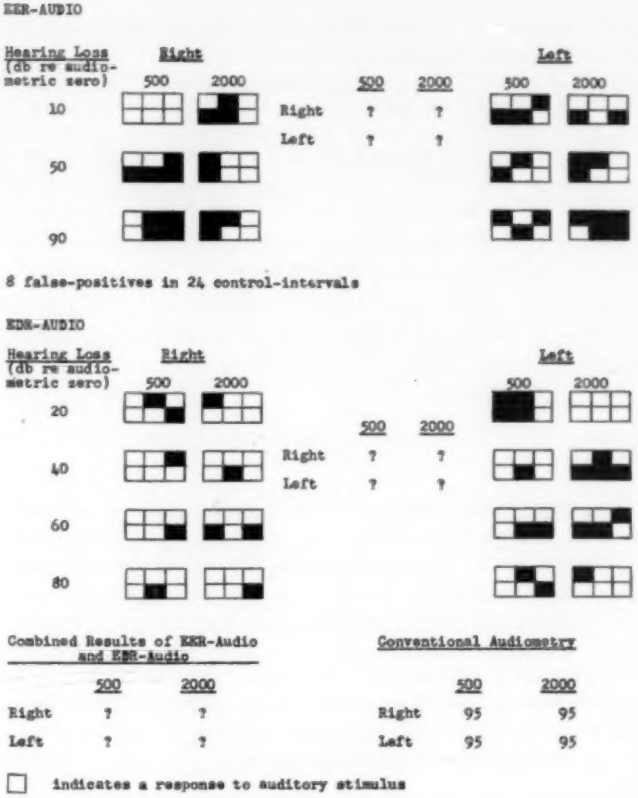

Fig. 10. Plot of responses from the EER-Audio and EDR-Audio for Subject G.

educational recommendations similar to the previous case (Case E).

G. 6 years old (see Fig. 10).

From the distribution of responses shown in Fig. 10, it is obvious that a hearing-loss based on the EER-Audio or EDR-Audio would have questionable validity. In addition,

the occurrence of a false-positive for every three controls in the EER-Audio requires five responses to six stimuli as evidence for hearing at a given stimulus-level. This criterion is met only at the 90 db. level for 2000 c.p.s. in the left ear.

There are slightly more EER and EDR to stimuli in the left ear than in the right. In the EER-Audio there is a small tendency for more responses to the more intense sounds than to the weaker sounds. Neither trend appears significant.

Conventional audiometry was relatively unsuccessful, and although the EER-Audio can be considered partially confirmative of the hearing-losses that were estimated, attempts to measure hearing-loss by electrophysiologic techniques were actually unsuccessful.

DISCUSSION.

The procedures outlined in this paper appear to satisfy some of the requirements for objectivity: the response-mechanism is not dependent upon the active cooperation of the subject, no special variation in procedure is required for each patient (although the intensity range can be compressed if desired), and hearing-losses can be estimated on the basis of a previously established criterion. The electrophysiologic tests are also independent in that they require no prior knowledge of a patient's auditory behavior and no knowledge of a patient's overt behavior during the test.

The objectivity and independence of the test does not compel the tester to ignore the overt behavior of the patient. This behavior can be recorded in a separate protocol (cf. example E in section on results) and then be related to the results of the electrophysiologic test. Likewise, the results of other attempts to measure hearing-losses through behavioral responses can be compared with the results of the electrophysiologic tests. We endorse the view[1,11,14] that electrophysiologic tests should supplement, not supplant, the conventional methods of measuring hearing-loss.

On the basis of our own clinical and experimental experience, we believe that many of the EDR we elicit are uncon-

ditioned; certainly all of the EER during sleep are unconditioned. We believe that we are able to maintain a relatively high rate of responses because of the variation in stimuli which is a consequence of the random schedule. When the stimuli are varied with respect to frequency, intensity and ear, each stimulus tends to be somewhat novel and thus acts as an unconditioned stimulus.

Because so little adaptation of the procedure is needed for each patient and because of the relative simplicity of the task of analysis, a tester need have little experience with electrophysiologic techniques to administer these tests successfully, providing that he has a good understanding of the general principles of clinical audiometry. Audiologists with virtually no previous experience with either the EER-Audio or EDR-Audio have already used these techniques successfully in our clinics.

The procedures described in this paper are not intended to be definitive or final, but rather to be a prototype for electrophysiologic procedures for determination of hearing-losses. Since the preparation of this paper, we have already tried certain modifications in the procedures. For instance, in the EDR-Audio we are testing the feasibility of using intensities corresponding to 20, 60 and 100 db. hearing-loss instead of the 20, 40, 60 and 80 db. presently used. This change may serve to reduce the time of the test and make it more likely to elicit responses from profoundly deaf children.

Much experimental work is needed to produce refinements that will insure more success with any particular procedure. Such factors as the optimum duration of the stimulus and the optimum length of time between successive stimuli need to be investigated, as well as such technical aspects as control of the level of sleep, most satisfactory location of electrodes, and automatic analysis of records.

The results we have obtained indicate that our procedures can be used practically. Although we have presented results of our tests on children, there is no reason to believe that these tests could not work just as satisfactorily with adults.

In fact, we do use a modified form of the EDR-Audio for *confirmation* of hearing-losses in adults.

Success of the EER-Audio and EDR-Audio, as outlined in this paper, does not appear to be dependent upon the particular kind of child or the nature of his auditory disorder. In the examples presented in the previous section, subject C is the only one who can be classified as an uncomplicated deaf child. Subjects A and D are aphasic children with hearing-losses. Subject B has a hearing-loss, and his problem in communication is complicated by a low intellectual capacity. Subjects E and F are considered deaf children, although they were referred for education with a diagnosis of some defect of the central nervous system which impeded normal development of language. Both of these children have serious behavioral problems which complicate their auditory difficulties. The child who was unsuccessfully tested (subject G) is classified educationally as aphasic, and has additional complications of a probable hearing-loss and emotional problems.

It has been our experience that there is no discrepancy or, at most, a minor discrepancy between the hearing-losses determined by procedures requiring overt responses and the hearing-losses determined by electrophysiologic techniques when the results of both kinds of tests are reasonably clear. Because of this agreement, we usually feel confident that clearly determined hearing-losses from the EER-Audio and EDR-Audio can be reported as representing a child's sensitivity to sound, even when the results of the more conventional techniques are too inconsistent to be corroborative. Occasionally there is an appreciable discrepancy between the findings from the EER-Audio and the EDR-Audio. In these instances, the EER-Audio usually indicates less hearing-loss.

We have no clear understanding at present of the anatomic structures or of the physiologic processes involved in the electrophysiologic responses to sound. Although a report may be given within ± 10 db. of the intensities believed to be a child's "threshold of hearing," the report does not tell us how the child ordinarily reacts to sound at or above his threshold. The findings merely state how much intensity is

necessary to stimulate the auditory nerve to make it deliver enough impulses to the central nervous system to initiate two particular kinds of responses. The thresholds reported, therefore, probably represent the minimum strength of sound to which a child is organically capable of responding.

When a child is tested by both the EER-Audio and EDR-Audio, there is a greater probability of obtaining a certain estimate of a small range (±10 db.) within which the hearing-loss lies than when only one of the tests is given. Nevertheless, when either test is successful, it is possible to obtain a range for the hearing-losses certain enough and narrow enough to give important support for making particular recommendations for special education.

Each test has advantages and disadvantages affecting its clinical practicality and desirability. The EER-Audio is less desirable than the EDR-Audio for the following reasons: *1.* Technically, the EER-Audio is more difficult to administer (control of sleep, multiplicity of wires, more channels for recording, etc.); *2.* When a child is asleep during the EER-Audio, naturally it is impossible for him to display his normal overt responses to sound; *3.* It is impractical to condition electroencephalic responses in a sleeping child, as one would condition electrodermal responses to sound in a child who is awake; *4.* It is more difficult to discern electroencephalic responses in an electroencephalogram than it is to discern electrodermal responses in an electrodermogram.

The EER-Audio has these advantages over the EDR-Audio: *1.* Because no conditioning is used with the EER-Audio, there is no disturbance of the child with electric shock or other annoying unconditioned stimulus; *2.* During the EDR-Audio, most young children are restless, and they squirm, partially in boredom during a long and uninteresting (to them) test. Very often they scream, kick and are generally disturbed because of the discomfort of the shock and because they are aware of the wires, phones, and the strange circumstances of the whole test-situation. During the EER-Audio the children are asleep (usually before the application of electrodes) and thus are unaware of and unfrightened by the test; *3.* Con-

sistent electrodermal responses are difficult to elicit in very young children.[7] According to Derbyshire,[4] electroencephalic responses to sound do not seem to be limited by age, and this seems to be true in our experience as well.

If only one test or the other were to be used for young children, we should favor the EER-Audio, primarily on the basis of the greater ease of handling of the children and because of the greater likelihood of eliciting responses. Reliability and narrowing of the range in which the hearing-loss lies could be achieved by successive EER-Audios. We suggest, however, that if it is at all feasible, both the EDR-Audio and EER-Audio be done on the same child.

SUMMARY.

In two separate tests, electroencephalic responses (child asleep) and electrodermal responses (child awake) to sound are used as indicators of "hearing." In both tests, tones of 500 c.p.s. and 2000 c.p.s. at different intensities are presented to either ear, according to a previously prepared, random schedule. The stimulus-levels in the first test correspond to 10, 50 and 90 db. hearing-loss; and in the second to 20, 40, 60 and 80 db. hearing-loss. The records are analyzed only after the tests have been completed and without knowledge, at the time of analysis, of the particular stimulus (or control) which corresponds to the ordinal number placed near each stimulus-mark. The combined results of both tests can yield an estimate of hearing-loss within ± 10 db. of thresholds determined from behavioral responses. Separately, either test yields less certain and precise estimates of hearing-loss.

Acknowledgments: The authors thank Dr. James O'Leary and members of his staff of the Division of Neurology and Electroencephalography, Washington University School of Medicine, for their help with the EEG equipment and with the administration of the sedatives; Dr. A. J. Derbyshire, Jr., Director of the EEG Department, Harper Hospital, Detroit, for his many helpful suggestions during the early stages of this study; and members of the Hearing Clinics and Research Department of Central Institute for the Deaf,

for their suggestions and help with the various clinical and technical aspects of this study.

REFERENCES.

1. Bordley, J. E.: An Evaluation of the Psychogalvanic Skin-resistance Technique in Audiometry. The Laryngoscope, 66:1162-1185, Sept., 1956.

2. Chaban, K. K., and Goldstein, R.: Relation Between EEG Pattern and Ease of Eliciting Electrodermal Responses. *Jour. Speech Hear. Disord.*, 22:651-661, Dec., 1957.

3. Derbyshire, A. J.: Personal communication.

4. Derbyshire, A. J.; Fraser, M. A.; McDermott, M., and Bridge, A.: Audiometric Measurements by Electroencephalography. *Electroenceph. Clin. Neurophysiol.*, 8:467-478, Aug., 1956.

5. Doerfler, L. G., and McClure, C. T.: The Measurement of Hearing Loss in Adults by Galvanic Skin Response. *Jour. Speech Hear. Disord.*, 19:184-189, June, 1954.

6. Gibbs, F. A., and Gibbs, E. L.: "Atlas of Electroencephalography," Vol. 1, Methodology and Controls. Addison-Wesley Press, Cambridge, Mass., 1950.

7. Goldstein, R.: Detection and Assessment of Auditory Disorders in Children Less Than Three Years Old. *Volta Rev.*, 57:215-219, May, 1955.

8. Goldstein, R., and Derbyshire, A. J.: Suggestions for Terms Applied to Electrophysiologic Tests of Hearing. *Jour. Speech Hear. Disord.*, 22:696-697, Dec., 1957.

9. Goldstein, R.; Ludwig, H., and Naunton, R. F.: Difficulty in Conditioning Galvanic Skin Responses; Its Possible Significance in Clinical Audiometry. *Acta Oto-laryngol.*, 44:67-77, Jan., 1954.

10. Mays, L. L.: A Device for Recording Skin Resistance with the Electroencephalograph Instrument. *Electroenceph. Clin. Neurophysiol.*, 5:313-316, May, 1953.

11. Rosen, J.: The Place of GSR Audiometry in Work with Young Children. *Volta Rev.*, 58:387-391, Nov., 1956.

12. Rosenblut, B.; Bilger, R. C., and Goldstein, R.: Electrophysiologic Responses to Sound as a Function of Intensity, EEG Pattern and Sex. *Jour. Speech, Hear. Disord.* In Press.

13. Stewart, K. C.: Some Basic Considerations in Applying the GSR Technique to the Measurement of Auditory Sensitivity. *Jour. Speech Hear. Disord.*, 19:174-183, June, 1954.

14. Statton, P., and Wishart, D. E. S.: Pure-tone Audiometry in Young Children; Psychogalvanic-skin-resistance and Peepshow. *Ann. Otol.*, 65: 511-534, June, 1956.

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EVALUATION OF THE ORTHOPEDIC SEPTO-COLUMELLAR SUTURE IN RHINOPLASTY.

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Boston, Mass.

The beginner in rhinoplasty is usually taught that essentially all nasal tips fall postoperatively after the septo-columellar sutures are removed and that it is, therefore, necessary to overcorrect or elevate the nasal tip on the septum in every case. In order to effect this, he raises the tip by means of the so-called orthopedic septo-columellar suture applied high on the septum and low on the columella, with the hope that the tip will eventually come down just right and that the postoperative esthetic effect will result in an attractive nose. In many cases it does just that—it comes down just right. Frequently, however, the tip falls down too much, resulting in a nose that is too long, a tip too round, and the cartilaginous dorsum of the septum too high; and there are rare occasions when the tip for some unaccountable reason remains too high. Too much, therefore, is left to chance, and under these conditions there is no control over the amount of pull and direction that the tip will take.

The teaching is fallacious. There are times when the orthopedic septo-columellar suture is definitely contraindicated, and under no circumstances should be used. At other times it is obligatory and must be employed, otherwise failure will surely result. All rhinoplasty surgeons agree that the orthopedic septo-columellar suture should not be used routinely in rhinoplasty.

When the feasibility of using the orthopedic suture is contemplated, it is helpful to divide nasal tips into two types: Type I, which possesses adequate projection with a sufficiently long columella; and Type II tip with inadequate projection,

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associated with a columella that is too short. The reason for dividing them in this manner is because each type requires a different treatment when the use of the orthopedic septo-columellar suture is considered.

The orthopedic suture should never be used in the Type I tip and is never indicated, providing the cartilaginous dorsum of the septum is lowered adequately at operation. I think this is of primary importance and should be emphasized. The orthopedic septo-columellar suture, on the other hand, is obligatory in Type II. Unless it is used in every tip of Type II the result will be an unsatisfactory rhinoplasty, even in the hands of the experienced surgeon.

TYPE I NASAL TIP.

This is the commonly encountered nasal tip, the one most frequently operated upon. It is the large oversized nose with a prominent nasal tip that possesses adequate projection. When the cartilaginous dorsum is left too high in such a nose, the judgment of the surgeon is usually at fault, and is the initial mistake leading to a train of errors. In order to create a straight profile at operation, the surgeon makes a second mistake by elevating the nasal tip to the level of the too high cartilaginous dorsum of the septum. This he accomplishes by means of the orthopedic septo-columellar suture applied high on the septum and low on the columellar. What is desirable is not an increase in the elevation of the nasal tip. Instead, the cartilaginous dorsum of the septum should be lowered to the level of the nasal tip. The technique for lowering the cartilaginous dorsum of the septum adequately has been described in a previous article.[3] The nasal tip, by means of this technique, stands up in its natural position unassisted and unsupported by artificial means. Under these conditions, it will retain its position postoperatively. Certainly, overcorrection of the tip on the septum by means of the orthopedic septo-columellar suture is contraindicated and should be condemned in every case of Type I tip. Light catgut sutures between the vestibular skin of the columella and nasal septum will suffice in approximating the columella to the septum.

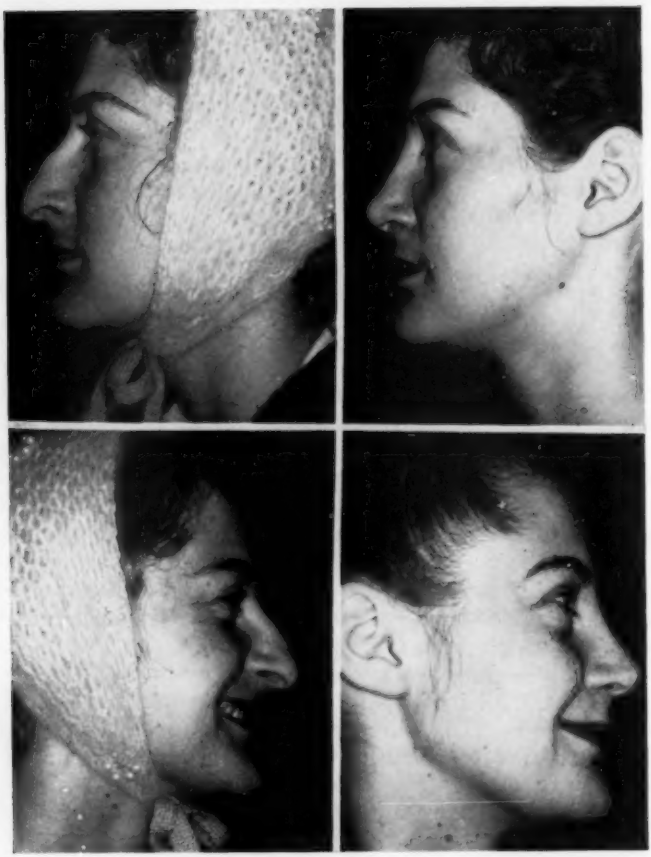

Fig. 1. Type I nasal tip.—Adequate projection of tip with sufficiently long columella. The orthopedic septo-columella suture is never indicated in Type I nasal tip.

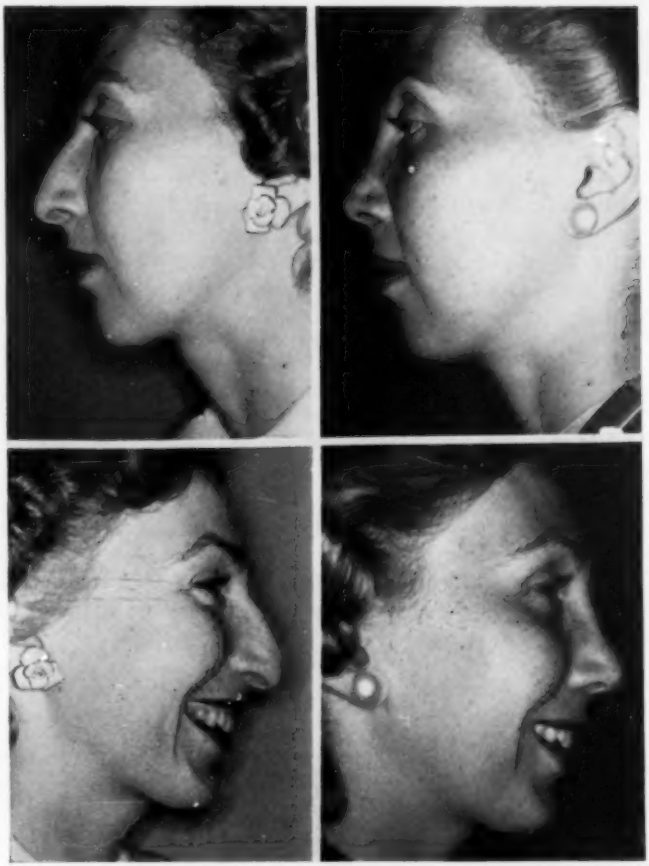

Fig. 2. Type II nasal tip.—Inadequate tip projection associated with a short columella. The orthopedic septo-columella suture is indicated in every case of Type II and should be used in conjunction with invagination and severing the depressor septi nasi muscle in the columella.

TYPE II.

The Type II nasal tip is totally different and far more difficult to correct than Type I. Bold, heroic treatment is obligatory. Overcorrection by means of the orthopedic septo-columellar suture is absolutely essential and must be resorted to in every case. This suture, however, applied in the conventional manner will fail to hold the tip in the elevated position unless it is supplemented by other means. In a previous article,[1] I advised invagination to insure the elevated position of the tip; and the lip-freeing technique[2] (cutting the depressor septi nasi muscle fibers in the columella) to prevent the lip muscles from pulling the tip down. With these additional methods of treatment, the tendency of the tip to return to its former low position is minimized and often eliminated.

An attempt should be made to lower the cartilaginous dorsum of the septum to the level of the nasal tip, as was done in Type I, but this is not always possible. There is a limitation to which we can lower the anterior surface of the septum. It should never be lowered less than 30 degrees in the nasofacial angle; otherwise the nose would be too small, the profile too low, and the tip too snubbed. For esthetic reasons, therefore, the tip must be raised above the level of the cartilaginous dorsum of the septum in every case of Type II and the orthopedic septo-columellar suture is the only means of doing so with a reasonable chance of success.

CONCLUSION.

When the orthopedic septo-columellar suture is contemplated in rhinoplasty, the first point to determine is the amount of projection of the nasal tip. Nasal tips can be profitably divided into two types: Type I with adequate projection; and Type II with inadequate projection.

The orthopedic septo-columellar suture is never indicated in any case of Type I nasal tip and will not be necessary, providing the cartilaginous dorsum of the septum is lowered adequately.

Overcorrection of the tip on the septum by means of the

orthopedic septo-columellar suture is obligatory in every case of Type II. In order to keep the tip in the elevated position, overcorrection must be combined with invagination,[1] and severing all the fibers of the depressor septi nasi muscle[2] in the columella to insure the permanent stability of the elevated tip.

REFERENCES.

1. Fred, G. B.: Nasal Tip in Rhinoplasty. *Ann. Otol., Rhinol. and Laryngol.*, 59:215, March, 1950.

2. Fred, G. B.: Role of Depressor Septi Nasi Muscle in Rhinoplasty. *A.M.A. Arch. Otolaryngol.*, 62:37, July, 1955.

3. Fred, G. B.: Post-operative Dropping of Nasal Tip After Rhinoplasty. *A.M.A. Arch. Otolaryngol.*, 67:177, Feb., 1958.

520 Beacon Street.

Permission for publication of these photographs has been secured from these patients.

MUMMIFIED EPIDERMAL CYST; CALCIFIED EPITHELIOMA OF MALHERBE.

Report of a Case.

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and
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The modern otolaryngologist is frequently confronted with tumors of the head and neck. An accurate diagnosis is always stressed, for such is important for clinical treatment as well as academic interest. The case herewith reported was recently seen in the Department of Otolaryngology and Maxillofacial Surgery, and in the Department of Dermatology at the State University Hospitals, Iowa City, Iowa. The exact nature of this tumor was not appreciated until a frozen section was done at the time of surgical excision.

CASE HISTORY.

The patient, a 13-year-old boy, was admitted to the State University Hospitals April 1, 1957. The chief complaint of the patient was a slowly growing tumor beneath the skin overlying the posterior aspect of the right mastoid process. The tumor was of two and one-half years' duration. There was no history of fever, cellulitis, suppuration, fluxuation, or breakdown of the tumor. Two weeks before admission the growth was incised by the referring physician and yielded several drops of blood. There was no regional lymphadenopathy. The patient lived on a farm and drank unpasteurized milk.

Physical Examination:

The patient was a husky young white male who appeared to be at the stated age of 13 years. He was neither acutely nor chronically ill. Oral temperature was 99.2° F.; pulse was 88/min., and BP was 110/70 mm. Hg. The tumor was firm and non-tender, and was overlying the right mastoid process (see Fig. 1). It was conical and measured 3.5 cm. at its rounded base and 2.0 cm. in height; it was freely moveable within the subcutaneous tissues. The overlying skin was reddish-brown and contained

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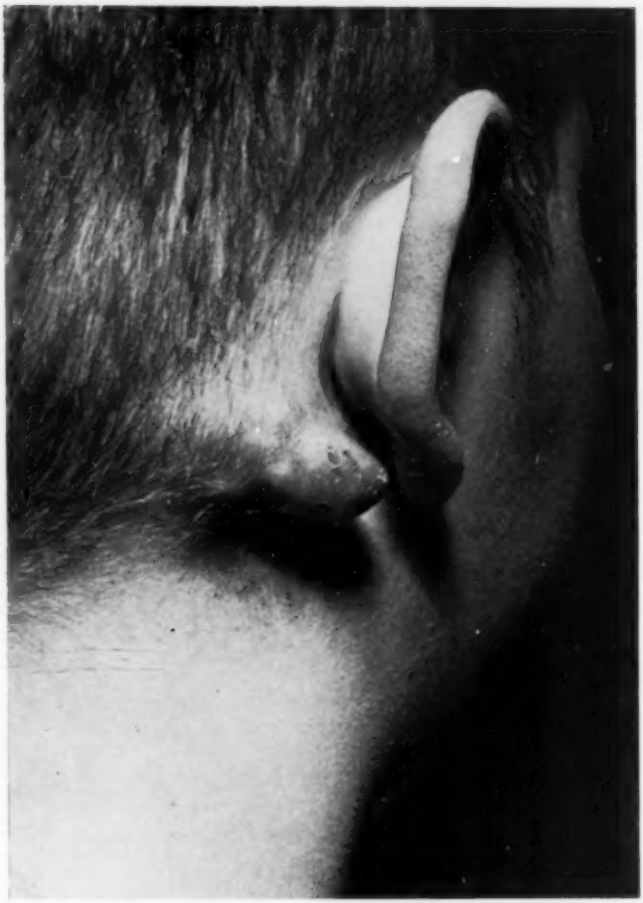

Fig. 1. Photograph showing the tumor overlying the right mastoid process of a 13-year-old boy.

three flecks of yellow-white material up to 0.1 cm. in diameter. The remainder of the physical examination was not remarkable. The ears, nose and throat were essentially normal; no change was noted in the faucial tonsils. The examination of the heart, lungs, abdomen, genitalia, and extremities was as usual.

Laboratory Studies: Complete blood count and urinalysis were within normal limits. The erythrocyte sedimentation rate was not elevated.

X-rays of the paranasal sinuses showed minimal thickening of the mucosa of the right antrum. An X-ray of the tumor was interpreted as showing no calcification; chest X-ray showed no change. Serological test for syphilis was normal. Intradermal tests for coccidiodomycosis, blastomycosis, and histoplasmosis were negative. Intradermal second strength P. P. D. test for tuberculosis was weakly positive.

Hospital Course:

The patient was seen in consultation by the Department of Dermatology, whose impression was a low grade malignancy of connective tissue origin. Excision was recommended. The preoperative diagnoses

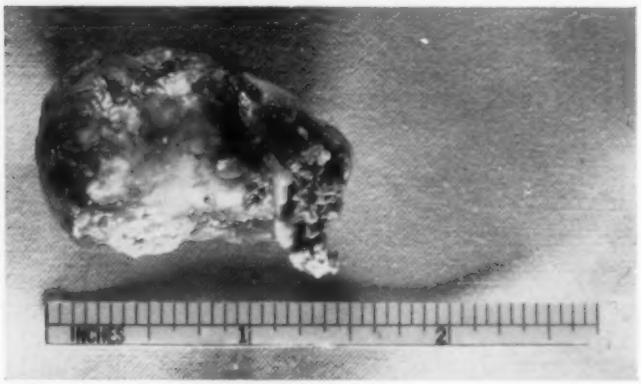

Fig. 2. Photograph of the oblong irregularly lobulated tumor. A portion of the closely adherent overlying skin is seen on the right.

included: 1. Low grade malignancy of connective tissue origin; 2. chronic granuloma, and 3. possible xanthoma. At the time of surgery the frozen section diagnosis was "Mummified Epidermal Cyst." The tumor was completely excised. A primary closure was done, and post-operative healing was uneventful.

Gross Description: The specimen consisted of an oblong, irregularly lobulated firm grey-brown tumor, measuring 4.0x2.5x2.0 cm. (see Fig. 2). An eliptical portion of overlying skin was closely adherent to the superior surface. Attached fragments of adipose tissue were scattered about the surface. On the cut surface, (see Fig. 3), the margins of the tumor were encapsulated by an intact covering of grey-white tissue, measuring 0.3 cm. The central portion was composed of yellow-grey compressed crumbly material which showed no particular pattern. No odor was discernible.

Microscopic Examination: The tumor was within the sub-cutaneous tissues (see Fig. 4). The stratified squamous epithelium of the overlying skin was well preserved and showed minimal acanthosis. Within the compact dermis there was an increase in collagen and fibrous connective tissue, and a sparse infiltration of lymphocytes.

The tumor was bordered by a thin layer of compact connective tissue,

representing a pseudo-capsule. Adjacent adipose tissue was present. The tumor was composed largely of masses of eosinophilic material which varied in size and shape. Some showed "ghosts" of former cells, whose appearance was that of having originally been stratified squamous epithelium. These cells had no remaining nucleus or visible cytoplasm, and only the cell walls were seen (see Fig. 5). Other eosinophilic masses were amorphous, showing no cellular structure. Within and about these eosinophilic masses there were minute foci of amorphous, deeply blue staining bits of calcium (see Fig. 5).

The stroma varied in composition, being loose in several areas but appearing compact in most areas. It was composed of strands of collagen and fibrous connective tissue. In some areas, an occasional fibroblast

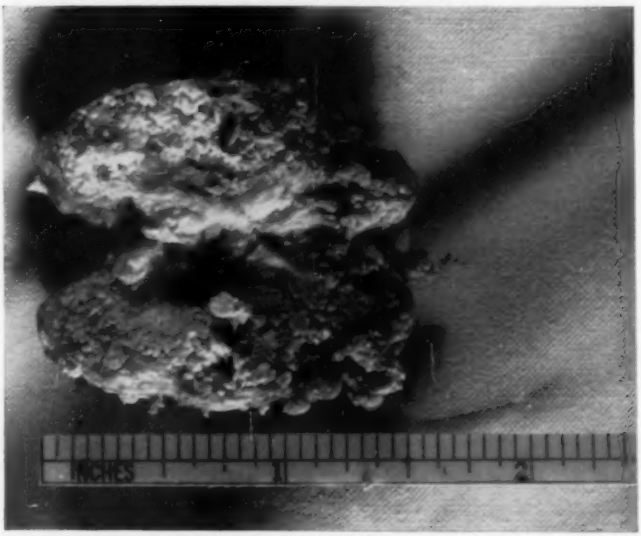

Fig. 3. Photograph of the cut surface of the tumor.

was seen. Scattered about the stroma there was keratin debris, about which there were several large foreign body giant cells and foci of lymphocytes (see Fig. 5).

The most interesting feature was the large groups of actively proliferating epithelial cells, which were intermingled within the areas of coagulative necrosis and amorphous keratin debris (see Fig. 6). These cells were closely packed and had a large pale blue vesicular nucleus containing one to two nucleoli (see Fig. 7). These cells resembled the basal cells of skin. The cytoplasm stained pale blue to light pink, and the cell borders tended to be indistinct. There were areas within these cell groups showing 4-5 mitoses per high power field (see Fig. 7). Two fields showed transformation of these cell groups into degenerating

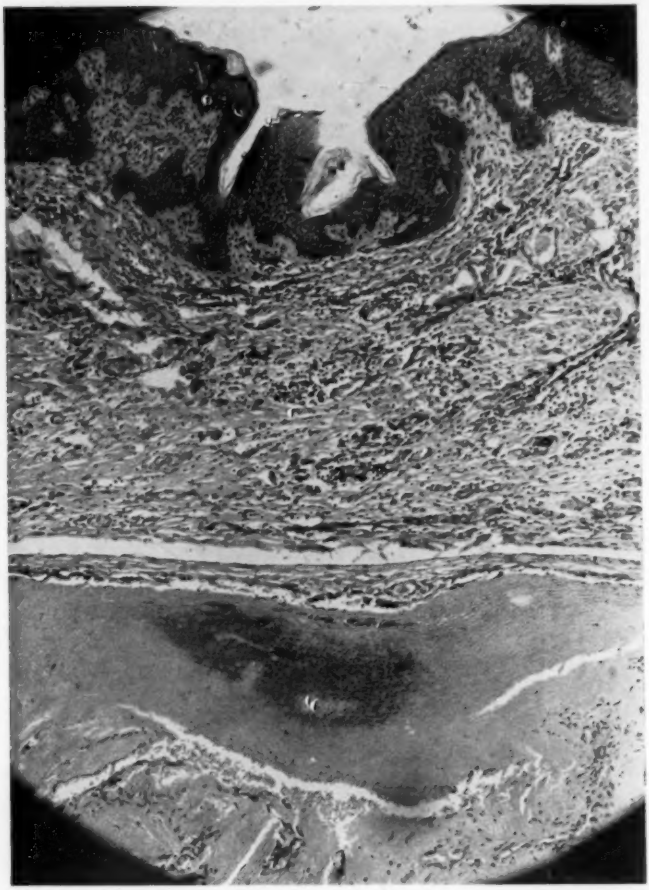

Fig. 4. Photomicrograph showing the margin of the tumor and the overlying skin—75X.

stratified squamous epithelium. In one focus there was dyskeratosis. Two small keratin pearls were present within one basal cell group.

DISCUSSION.

This tumor was first described in 1880 by Malherbe (as cited by Lever[1]). It is unfortunate that Malherbe used the

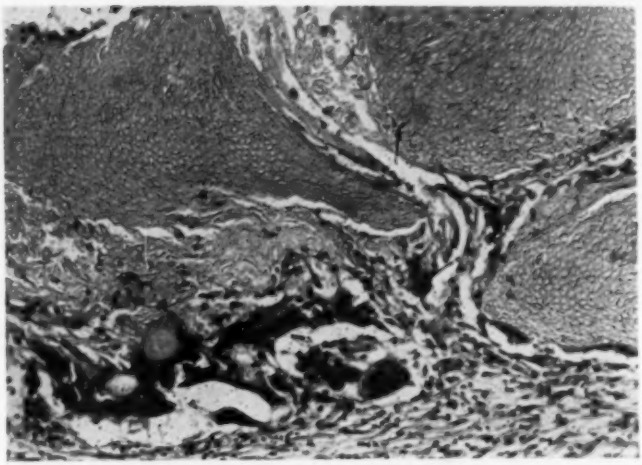

Fig. 5. Photomicrograph showing three groups of "ghost cells" (degenerating stratified squamous epithelium). Within the stroma there are foreign body giant cells adjacent to keratin debris and flecks of calcium—300X.

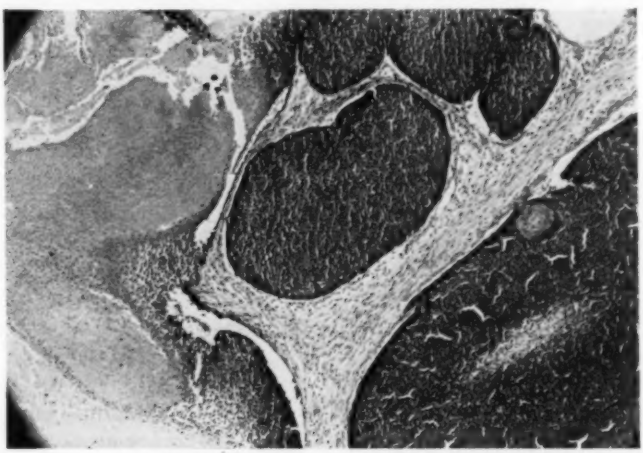

Fig. 6. Photomicrograph showing groups of actively proliferating basal cell type epithelial cells. The transformation of these basophilic cells into the shadow cells can be seen—100X.

term "calcifying epithelioma" in describing the lesion, for it neither metastasizes nor locally invades. In 1933, Chin Kuang-Yu[2] reviewed the world medical literature on the subject; he postulated that perhaps these tumors arose from embryonic rest cells. King,[3] in 1947, introduced the name of "Mummified Epidermal Cyst," for he believed, as do the authors, that the tumor is related to epidermal cysts of the skin. In cases where there is a history of previous trauma to the skin overlying the tumor, there is little doubt as to the

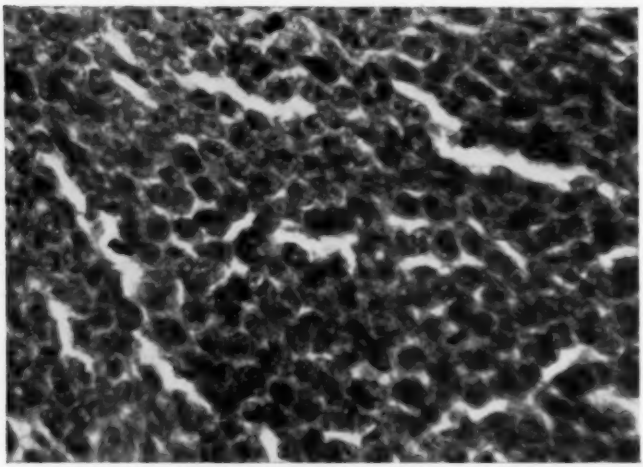

Fig. 7. Photomicrograph showing the basal cell like epithelial component of the tumor. Seven mitoses are seen in this field—600X.

relationship of the tumor to an epithelial cyst or epithelial implant.[3]

Various reports list different incidences for this tumor. In the earlier report the incidence was sometimes one in ten thousand surgical specimens,[2] and in the later reports the incidence has been sometimes one in two thousand surgical specimens.[3] There is apparently no difference in the incidence of the tumor according to sexes. The tumor may appear any time between the ages of two years to 80 years,

but it has recently been reported to be more common in childhood.[4]

The growth may occur on the scalp, face, back, arm or thigh; but it is most common about the head and neck.[5] The lesion is slow growing, and the majority have been present for an average of three years before being removed.[6] The tumor is attached to the overlying skin, but it is freely moveable within the underlying tissues. Rarely, there may be ulceration of the overlying skin.[5] The tumor is non-tender and usually asymptomatic. It is rubbery to firm, to palpation. Recommended treatment is surgical excision. The most common preoperative diagnosis is a sebaceous cyst.[4]

Grossly, the tumor usually measures between 1.0 to 4.0 cm. in greatest dimension, although some have been reported up to 7 cm.[3] The surface is grey-white, smooth to finely lobulated, and has particles of adipose tissue adherent to its thin capsule of fibrous connective tissue. At times, the capsule is not complete. The firm tumor often cuts with some difficulty because of the bits of calcium which are almost always present within its stroma.[2] The cut surface is light tan to yellow brown, and varies from smooth to granular to crumbly.[3] Microscopically, there are two basic cell types: the "basophilic cells" or basal cell type, and the "shadow cells" (mummified epithelial cells) or squamous cell type.[1,3]

The varying sized irregularly shaped groups of mummified or shadow cells are examples of coagulative necrosis, for although the cell borders are distinct, the nuclei and cytoplasm have undergone necrosis and do not stain. In occasional foci the squamous type epithelial cells may show cornification, while in other areas this epithelial component may show dyskeratosis. In other portions the necrosis may have progressed so that only amorphous hyalin debris remains.

The basophilic cells, or basal-cell-like epithelial component, are also found in varying sized groups. There is no pallisading about the periphery of these groups, thereby differentiating them from basal cell tumors. These cells have rounded to oblong prominent deep blue vesicular nuclei with one to two nucleoli. The cytoplasm is scant and light blue with ill-

defined cell borders. Mitoses are not unusual. In areas, one may see the transformation of the basophilic cells into the shadow cells.[1]

Within the stroma there are varying scattered flecks of calcium, which stain blue-black with hemotoxylin and eosin. There are also foci of foreign body giant cells about the keratinous debri. The bulk of the stroma is composed of collagen and fibrous connective tissue.

Although the microscopic picture of this tumor is easily recognized, a calcified epidermal cyst should be considered in the differential diagnosis. Lever[1] states that a calcifying epithelioma of Malherbe differs from a calcified epidermal cyst, in that the latter does not contain the basophilic cells or basal cell type. This distinction, however, is not universally recognized.[3]

SUMMARY.

1. Mummified epithelial cysts (calcified epitheliomas of Malherbe) are relatively common tumors of the head and neck.

2. This tumor may occur in any age group, but it is most common in children.

3. The lesion is slow growing, firm, sub-cutaneous, and freely moveable.

4. The lesion has two basic cells types; the basophilic or basal cell type, and the shadow or mummified squamous cell type.

5. The tumor does not metastasize and does not locally invade.

6. The treatment of choice is surgical excision.

BIBLIOGRAPHY.

1. Lever, W. F.: "Histopathology of the Skin," pp. 316-320, J. B. Lippincott Co., Philadelphia, 1949.

2. Chin Kuang-Yu, Calcified Epithelioma of Skin. *Amer. Jour. Path.*, 9:497-524, July, 1933.

3. King, L. S.: Mummified Epidermal Cysts (So-Called "Calcified Epitheliomas"). *Amer. Jour Path.*, 23:29-43, Jan., 1947.

4. Cawley, E. P., and Wheeler, C. E.: Calcifying Epithelioma, with Emphasis on Its Occurrence in Children. *South. Med. Jour.*, 48:898-901. Sept., 1955.

5. Castigliano, S. G., and Rominger, C. J.: Benign Calcifying Epithelioma of Skin. *Arch. Derm. and Syph.*, 70:590-600, Nov., 1954.

6. Highman, B., and Ogden, G. E.: Calcified Epithelioma. *Arch. Path.*, 37-38:169-174, March, 1944.

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Second Vice-Presidente: Dr. Carlos Arango Carbone.
Secretario: Dr. Maria Esther Villalaz.
Tesorero: Dr. Ramòn Crespo.

SOCIEDADE PORTUGUESA DE OTORRINOLARINGOLOGIA E DE BRONCO-ESOFAGOLOGIA.

Presidente: Dr. Alberto Luis De Mendonca.
Vice-Presidente: Dr. Jaime de Magalhaes.
1.° Secretario: Dr. Antonio da Costa Quinta.
2.° Secretario: Dr. Albano Coelho.
Tesoureiro: Dr. Jose Antonio de Campos Henriques.
Vogais: Dr. Teofilo Esquivel.
Dr. Antonio Cancela de Amorim.
Sede: Avenida da Liberdade, 65, 1°, Lisboa.

SOCIETY OF MILITARY OTOLARYNGOLOGISTS.

President: Lt. Col. Stanley H. Bear, USAF (MC), USAF Hospital, Maxwell (Air University), Maxwell Air Force Base, Ala.
Secretary-Treasurer: Capt. Maurice Schiff, MC, USN, U. S. Naval Hospital, Oakland, Calif.
Meeting: October 14, 1958, Palmer House, Chicago, Ill.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. James H. Gressette, Orangeburg, S. C.
Vice-President: Dr. Robert P. Jeanes, Easley, S. C.
Secretary-Treasurer: Dr. Roderick Macdonald, 333 East Main St., Rock Hill, S. Car.
Meeting:

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Dr. V. Eugene Holcombe, Charleston, W. Va.
Chairman-Elect: Dr. G. Slaughter Fitz-Hugh, Charlottesville, Va.
Vice-Chairman: Dr. George M. Haik, New Orleans, La.
Secretary: Dr. Mercer G. Lynch, New Orleans, La.

VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Benjamin Sheppard, 301 Medical Arts Building, Richmond, Virginia.
President-Elect: Dr. Emanuel U. Wallerstein, Professional Building, Richmond, Virginia.
Vice-President: Dr. Calvin T. Burton, Medical Arts Building, Roanoke, Virginia.
Secretary-Treasurer: Dr. Maynard P. Smith, 600 Professional Building, Richmond, Virginia.
Meeting:

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. James K. Stewart, Wheeling, W. Va.
Secretary-Treasurer: Dr. Frederick C. Reel, Charleston, W. Va.
Annual Meeting: